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ECHINOCOCCUS ALVEOLARIS, WITH REPORT OF AN AUSTRALIAN CASE.

By HAROLD DEW.

[From the Department of Surgery, University of Sydney.]

THE case here reported is of fundamental importance to all students of hydatid disease in that it is the first authentic record of this peculiar form of human echinococcal infestation in Australia. Also, it is, with the exception of the case recorded by Wernicke[1] in South America (of doubtful authenticity because the birthplace of the patient was not known with certainty), the first case recorded in a country outside the rather narrow geographical distribution of the disease in Europe. It therefore has an important bearing on the two doctrines that have held sway in Europe during the last fifty years concerning the true nature of the lesion. Advantage has been taken in this paper to review briefly the history of the rise of the two schools of thought and the various arguments, pathological, experimental and geographical, that have been advanced from time to time by those supporting one or other view. In addition, the significance of this case is emphasized and certain conclusions are drawn from it.

CASE HISTORY.

The patient, S.L., a male, aged sixty-seven, had spent his whole lifetime in Victoria and Tasmania, being employed until the age of sixty as a labourer and foreman on railway construction. This entailed working on many occasions in country districts of both of these States, often under unhygienic conditions. He had had no previous serious illness and until five years before admission to the Melbourne Hospital (September 26, 1928) had regarded himself as particularly healthy. From that time he had had occasional attacks of flatulence which had been regarded as dyspeptic in origin and had been more or less disregarded. Three months before admission he felt out of sorts and had been a little "liverish," with occasional attacks of pain in the back and epigastrium, but this was quite relieved by counter-irritation and mild purgation. Three weeks before admission he noticed some swelling of the ankles, some diminution in the quantity of urine, but no difficulty or frequency of micturition, although some suprapubic discomfort

was present. During this period his appetite remained fairly good, he had flatulence, but no nausea or vomiting, and there was no interference with bowel function.

A fortnight later he noticed that he had some yellowness of the skin and conjunctivæ, and that his abdomen seemed to be a little larger. At the same time, he became easily tired and had some shortness of breath on exertion. He stated on admission that he had noticed his urine had been darker in colour for about two weeks, and thought that his abdomen had really been increasing in size for several weeks. On examination he was deeply jaundiced, but was for his age well preserved and well nourished. The heart and lungs appeared normal, although there was some encroachment on the right base by the hepatic dulness. The abdomen showed enlarged parietal veins, gross swelling and obliteration of the umbilicus, but no *caput medusæ*. The hepatic dulness extended three to four finger-breadths below the costal margin and the edge of the liver felt hard and nodular, while in the region of the gall bladder was a larger hard mass extending almost to the iliac crest. The hepatic dulness also extended as far up as the fourth rib in the mid-axillary line. Gross ascites was present. The spleen was not palpable and, as far as could be detected, the other abdominal organs were normal. There was no œdema of the lower extremities and the other systems showed no abnormality.

He was at first regarded as suffering from carcinoma of the liver or gall bladder, although he had not lost weight and appeared relatively well for a patient with malignant disease. During the first week in hospital the jaundice increased a little, but after three weeks it had subsided almost completely. I first saw the patient at this stage, and suggested that the diagnosis was probably hydatid disease of the liver with possibly choleperitoneum from a quiet leak, and suggested that several special examinations be carried out.

Paracentesis abdomini was performed and bile stained fluid, which, however, contained no hydatid elements, was drawn off. The Casoni intradermal test was performed during the jaundiced period, but gave no reaction. The hydatid complement fixation test, ordinary hydatid antigen being used, gave a strongly positive reaction by both the warm and the ice-box methods, while the Wassermann test gave no reaction. The Van den Bergh test showed an immediate positive reaction. A special radiographic investigation was carried out, and showed limited movements of the right diaphragm, which was also raised, while a slightly curved shadow 5·0 centimetres by 0·3 centimetre (two inches by one-eighth inch) thick was demonstrated below the posterior part of the diaphragm, and was regarded as evidence of a partly calcified cyst wall. Examination of the fæces on two occasions for hydatid membrane revealed nothing abnormal.

The diagnosis of hydatid disease being apparently confirmed, on December 8, 1928, I explored the liver by the anterior route through the rectus muscle, under ether anæsthesia induced by the open method. After evacuating a very large quantity of bile stained ascitic fluid, the liver was found to be grossly enlarged and to contain in the right lateral and upper quadrant a peculiar hard nodular tumour, which at first closely resembled carcinoma. The gall bladder was enormously distended and a large gall stone could be felt in the cystic duct. After close inspection the lesion was considered to be a peculiar calcified hydatid cyst, and it was incised laterally to the gall bladder, when hydatid *débris*, calcareous and caseous material and bile stained fluid under pressure escaped. This caused diminution in the size of the gall bladder swelling, and the hydatid cyst, which was found to be multicystic with much central degeneration, was evacuated as far as possible, a communication with the gall bladder being discovered during this process. After carefully packing off the cyst, the gall bladder was opened and much *débris* and six large gall stones, including one in the cystic duct, were removed. Drainage tubes were inserted into the gall bladder, the cavity of the hydatid and into the subhepatic pouch through a stab wound.

The patient suffered from some post-operative shock, but rapidly improved. After forty-eight hours, however, the drainage from all tubes increased greatly, his temperature rose, his abdomen became very distended and he gradually sank

and died on the sixth day after operation in a condition resembling that of suppurative cholangitis and cholæmia.

Pathological Findings at Autopsy.

The peritoneal cavity contained some turbid bile stained fluid and some *débris*. The liver, which was removed entire, contained a very large multicystic hydatid tumour, the central part of which was evacuated and contained nearly two pints of bile stained *débris*. This *débris* was composed of caseous material,

FIGURE 1. Coronal section of liver showing central cavitation, thick adventitia above, adhesion to diaphragm, multicystic lesion in periphery, hypertrophy of left lobe of the liver and drainage tube in the gall bladder.

bile stained pus, broken down tissue, crumpled hydatid membrane and three small rounded cysts, resembling ordinary daughter cysts except that they had very thin clear walls. The upper part of the adventitia was adherent to the diaphragm, while the main cavity had a ragged trabeculated lining and communicated with the gall bladder. The latter was thick walled, almost gangrenous in places, and contained several small calculi and some *débris*. The common bile duct also contained some hydatid *débris*, but was not occluded. On the under surface of the liver the *porta hepatis* was found to be surrounded by several small subperitoneal extensions of the multicystic process. The largest of these measured 2·0 centimetres (four-fifths of an inch) in diameter.

The other organs seemed normal for the age and no hydatid disease was found elsewhere. Death was due to infected hydatid cyst of the liver, cholangitis and general peritonitis.

Pathological Nature.

The liver was placed in formalin preservative and, after hardening, the peripheral portions of the lesion were dissected free of adhesions and fat and the whole sectioned in the coronal plane (see Figure I).

This showed the right lobe of the liver almost completely occupied by a peculiar multiloculated cystic mass, the centre of which had undergone cavitation, most of the *débris* having been previously evacuated. The gall bladder was greatly enlarged, its wall thickened and it contained a few small gall stones. The left lobe of the liver was somewhat hypertrophied and showed a condition of nutmeg

FIGURE II. Part of the lesion showing intermediate sclerotic zone. × 3.

liver with some bile staining. The lesion was undoubtedly hydatid in nature and three separate zones could be detected. The central zone comprised the central cavity, which extended up to the diaphragm, which in turn was adherent to the thickened sclerotic and avascular adventitia, the latter being calcareous in part. The greater part of the cavity was lined with ragged seminecrotic, bile stained tissue, in the interstices of which irregularly folded pieces of hydatid membrane were present. The intermediate zone was 0·5 centimetre to 2·5 centimetres wide and was mainly composed of dense sclerotic fibrotic tissue surrounding multitudes of small cystic spaces which contained gelatinous hydatid membrane, showing various degrees of degeneration. This layer was thickest in the inferior part of the cyst, where it extended down to and apparently involved the wall of the gall bladder. The size of the cavities in this zone varied from a centimetre in diameter to others which were just visible to the naked eye. Many of the cavities contained bile stained *débris*, crumpled hyalin membrane or caseous-like material. Many portions of this zone showed a preponderance of dense fibrotic tissue over parasitic tissue (see Figure II)

The third zone occupied the periphery, where the lesion was juxtahepatic, the right lateral portion being almost entirely sclerotic. This zone showed infiltration and invasion of the hepatic tissue by a great number of what appeared to be small cysts, all containing hydatid hyalin material. A few of these cavities measured 1·5 centimetres in diameter, but the majority were very small, one millimetre or less in diameter. There was no definite adventitious capsule limiting the growth of the process, although there seemed to be an attempt to limit the growth of many of the cystic spaces, and many of the small spaces showed dense surrounding sclerotic tissue and a great deal of degenerative change within. A

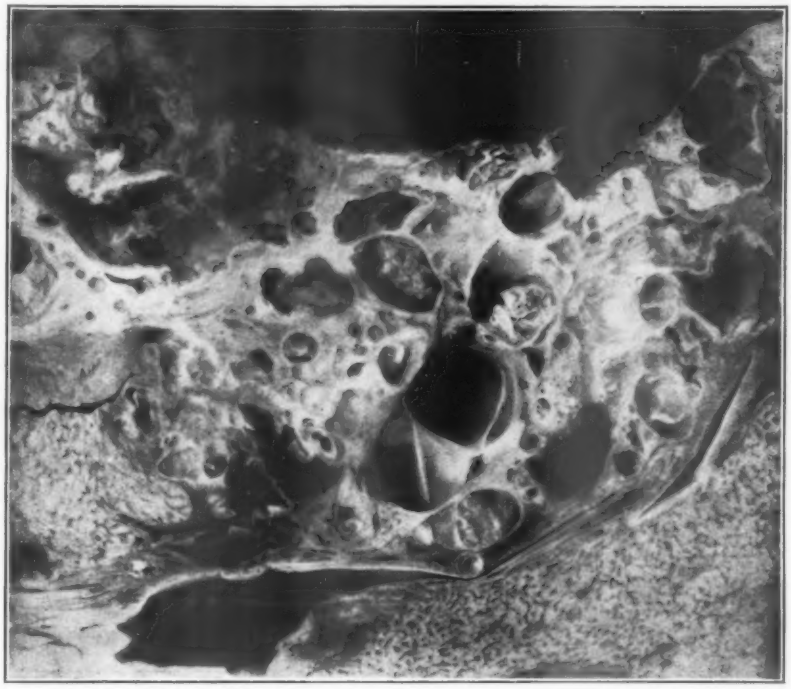

FIGURE III. Periphery of the lesion showing juxtahepatic portion, cystic infiltration of liver and some pressure on a large hepatic vein. × 3.

few of the larger cystic spaces were filled by clear fluid, but in none could scolices or their remains in the form of hooklets be detected (see Figure III). In the main cavity three small thin-walled cysts were lying free. These were all small, the largest being one centimetre in diameter, and although they resembled daughter cysts, it is probable that as none were noticed at operation, they had become secondarily extruded in an intact state out of the somewhat traumatized wall of the central cavity. None of them contained any scolices.

On the inferior and posterior aspect of the liver a dissection of the portal fissure disclosed seven projecting cysts under the peritoneum and fat of the region (see Figure IV). These were dissected clear and several examined after puncture and section. It was found that they were rather larger on the average than

the cystic spaces in the main lesion, the largest being two centimetres in diameter. They, too, were all sterile, and two of them contained bile stained caseous *débris*. Two others had caused much narrowing of the inferior *vena cava* without, however, actually invading its wall. On examination of the portal vein it was found to be thrombosed completely over a distance of three centimetres, owing, apparently, to the pressure of the hydatid infiltration behind it. This offered a complete explanation of the generalized ascites which had been present for some time. The common bile duct was somewhat larger than normal, but showed no trace of having suffered from the effect of external pressure.

FIGURE IV. Postero-inferior aspect of the lesion showing the subperitoneal extensions of the cystic process around the portal fissure.

In those places where the parasite had involved the diaphragm and the right peritoneal surface of the liver, its invasive tendency seemed to have been at once limited by the development of protective fibrous tissue. Only where it had actually ruptured into the gall bladder was there any true invasion of the extrahepatic tissues. Even here it would appear that the rupture was a matter of slowly developing pressure necrosis of the gall bladder wall and not a true parasitic infiltration. It would seem then that the parasite showed a striking predilection for hepatic tissue and that its invasive power was definitely limited. In the gall bladder there were several large gall stones, several of which showed hyalin material as a nucleus. This makes it certain that the actual leakage into the gall bladder took place long before the onset of clinical symptoms, and that it was probably a very gradual process.

NATURE OF THE LESION.

There was no doubt that the lesion was unique as far as Australian experience went, and it was regarded as an example of alveolar hydatid disease. Microscopical sections were taken from many parts, a great number of blocks being prepared and sectioned. Specimens were also taken and with photographs forwarded for an opinion to Dr. F. Dévé, of Rouen, who has made a special study of this particular branch of hydatid disease. He confirmed the diagnosis of *Echinococcus alveolaris*.

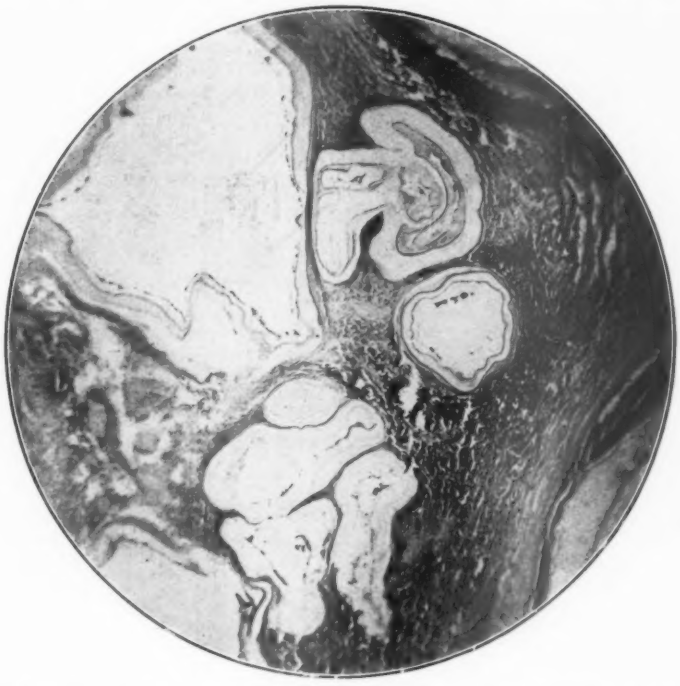

FIGURE V. Microscopical section showing some of the cystic spaces of the intermediate zone. Note the typical two-layered hydatid cysts, some collapse and degeneration, and the absence of systematic cellular reaction in the somewhat acellular, avascular, surrounding adventitial tissue. × 45.

The multicystic arrangement of the lesion, the lack of definite outline, the infiltration of the surrounding hepatic tissues, while cavitation and breaking down of the central portion proceeded, the irregular periparasitic fibrosis, the small, often microscopical size of the vesicles in the advancing edge and the size and site of the lesion were all classical. Microscopically the lesion showed varying pictures. In the areas where the cysts were easily visible to the naked eye, they showed an arrange-

ment not unlike that seen in some cases of *Echinococcus multilocularis*, so commonly seen in the liver of the ox and rather less commonly in the sheep (see Figure V).

The tissue immediately surrounding these small spaces did not, however, show the trizonal cellular arrangement so typically seen in the cysts of the ox. There seemed little doubt that in this case the parasitic tissues had become effete, and that the parasite was (if not actually undergoing degenerative changes) just managing to survive. The laminated membrane seemed to be poorly developed in comparison with the typical hydatid cyst and, as is common in this disease, the parasite seemed to be sterile. In spite of careful search by microscopical examination after centrifuging as much fluid as could be obtained from a great number of the spaces, no scolices could be found. Sections from near the central zone showed almost uniformly degenerative changes in the parasitic tissues, but not, as would be expected, any phagocytosis of the *débris*. The adventitious tissues surrounding these spaces were relatively acellular and avascular and showed much sclerosis. There seemed to be evidence that while the lesion was slowly progressing at the periphery, the fibrosis induced around the older part of the lesion had produced so much interference with the blood supply that the toxins produced were able to produce actual necrosis and death of the more central portions.

Sections from the periphery where the cystic spaces were very small or microscopical showed that the parasite was still actively invading the hepatic lobules. This invasion was brought about by active prolongations of both layers of the parasite, although in some of these the hyalin layer was deficient or missing, as has been described by Dévé as characteristic of the lesion. Surrounding these processes the cellular reaction of the host was extremely active (see Figures VIA, VIB, VIC and VID).

This reaction was composed of an inner zone of eosinophile and polymorphonuclear cells surrounded by radially arranged endothelial cells and fibroblasts. This arrangement was very similar to that seen in the reaction during the early stage of development of the ordinary hydatid parasite from the second to the twelfth week, that is, until the parasite has elaborated a relatively efficient laminated membrane. The diverticula on section at first gave the impression of being distinct and separate microscopical cysts, but serial sections showed that they usually, if not always, communicated with each other and any separate cysts present were probably due to secondary closure, resembling rather the mode of production of separate cysts in ordinary hydatid of bone. This was verified by making a wax model by building up the serial sections. In this lesion it appears that the parasitic extension into the host tissues takes place by means of multiple microscopical outgrowths, which grow persistently in spite of the cellular reaction of the host. The presence of many eosinophile and other leucocytic cells seemed to be evidence of leakage of toxin from the parasite, and this possibility was suggested too by the presence of apparently naked germinal nucleated parasitic membrane in some processes. It is probable that the hyalin laminated

FIGURE VIA.

FIGURE VIB.

FIGURE VIC.

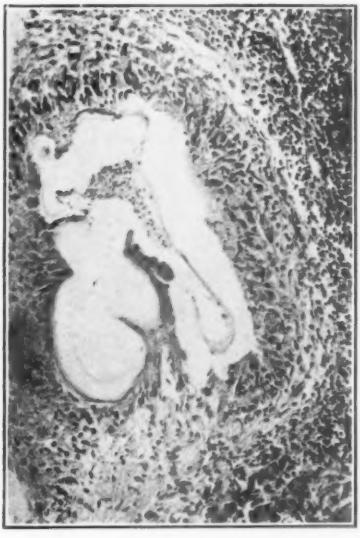

FIGURE VID.

Series of four serial sections (every sixth section) of actively growing edge of lesion, showing mode of growth, active cellular reaction of the host tissues, and the formation of diverticula. This also shows the microscopical size of the parasitic processes and the poor development of the laminated membrane. × 140.

membrane here was wanting, deficient or effete, and it may be that therein lies the explanation of the uncontrolled parasitic growth and the persistent toxic effects on the surrounding cells of the host. Nowhere were any appearances even remotely suggesting the "embryonic" growths described by Melnikoff[2] seen.

In all essential respects this lesion corresponded with the classical description of Dévé,[3] namely:

1. Innumerable, irregular and minute cavities containing crumpled and discoloured gelatinous hyalin material all enclosed in a dense, avascular, fibrotic adventitious tissue, which often shows some calcification.

2. The cavities rarely exceed the size of a hazel nut; there is, as a rule, little hydatid fluid present in them; scolices are often absent and the lesion as a whole after fixation looks rather like a sponge.

3. No trace of encapsulation; irregular and indefinite outlines, the lesion insidiously infiltrating the surrounding tissues.

4. Central necrosis and cavitation of the central portions of the lesion, the resultant brownish bile stained fluid containing hyalin, caseous and cellular *débris*.

Although the lesion here reported does not illustrate them, there are other characteristics upon which great emphasis has been laid. There is undoubtedly a strong tendency in some cases towards invasion of the regional lymph glands and for blood-borne metastases to occur in other organs. It is important, too, to note that not only in these metastases, but in the cases in which the lesion is primary in other organs, such as the brain, spleen or lung, there is a surprising uniformity in both the macroscopical and microscopical appearances. According to Dévé, this is also true of the lesions recorded, irrespective of the locality where they have been encountered.

It does, however, differ in some particulars from the classical lesions and presents several interesting features, a discussion of which will be postponed till later. Before considering the significance of the discovery of this disease in Australia, a short review of the subject is presented.

HISTORICAL.

Before the year 1855 various European pathologists had observed a curious hepatic lesion which, owing to its infiltrative characteristics, closely resembled malignant disease. It was generally regarded as a variety of colloid cancer until in the above year Virchow[4] first recognized its helminthic nature and named it ulcerating multilocular hydatid of the liver. Since that time many opinions have been expressed as to the true nature of this hydatid process, and clinicians, pathologists, helminthologists and veterinarians have all been drawn into the controversy. All were well acquainted with the clinical and pathological manifestations of the common universally distributed hydatid disease, but this lesion bore so little resemblance to this that the question of the existence of two distinct parasites was soon raised. Virchow[4]

from the beginning upheld the unicist doctrine that the parasite was a "*Formvarietät*" and suggested that the peculiar arrangement of the vesicles and parasitic tissues generally was due to their development and growth along hepatic lymphatic channels. Küchenmeister,[5] Leuckart[6] and others also held similar views and, although they varied from each other somewhat in minor details, were strong protagonists of what has been called the "*Kanaltheorie*" of growth. It was very soon noticed that this particular form of the disease apparently had a very narrow geographical distribution, being practically confined to the more or less mountainous central states of Europe, whilst it seemed to be unknown in those countries like Iceland, Algeria and Australia, where the disease in its usual form was so common.

In 1875 Morin,[7] using the geographical argument, actively championed the view that there must be two distinct parasites and from this time a dualistic school of thought rapidly rose. Many feeding experiments were carried out in order to settle the vexed question, but unfortunately all of these must be regarded as inconclusive in their results. The literature of the subject has grown enormously and is extremely interesting, consisting as it does largely of a series of arguments on one side or the other, followed in a year or two with equally ably stated counter-arguments. This controversy between the dualists and unicists is still going on and the reader is directed to the excellent paper of F. Dévé[3] given before the First International Congress of Comparative Pathology at Paris in 1912 for the full story.

Dévé entered the lists in favour of the dualists in 1903, but has since that time rather modified his opinions. With the recognition of the disease in parts of the Russian Empire, Russian pathologists entered the controversy and although the earlier observers were dualists, the later ones favour the unicist doctrine. More recently, too, observers in South America have brought forward fresh evidence which they believe supports the unicists. The writer[8] in 1925, basing his deductions partly on analogy and partly on a study of the pleomorphism of hydatid disease as seen in the common domesticated animals, sided with the unicists in believing that there was nothing as yet demonstrated in the pathology of any hydatid lesion that could not be adequately explained by the occurrence of parasitic variation.

In spite of all the observations that have been made and of all the various theories put forward, the question is still undecided; but it is of interest to consider shortly the various types of evidence which have been put forward from time to time by one or other side.

GEOGRAPHICAL DISTRIBUTION.

It was the apparently narrow distribution of the alveolar disease that first suggested the obvious explanation that there may be two distinct parasites. The disease is particularly common in the mountainous countries of Central Europe—South Germany, the Tyrol, Bavaria and Serbia. In these countries one finds in the pathological institutes

almost as many examples of alveolar hydatid as of the ordinary disease. Schmidt[9] analysed 51 cases of hydatid disease occurring in all the Munich hospitals up to 1889 and found that almost half the cases were of the alveolar form. It is important to note in passing, however, that the total number of cases he recorded from these countries, which are fairly thickly populated, taken, no doubt, from an enormous number of total patients admitted to hospital, is very small, so that probably the total percentage incidence of hydatid disease in these people is much smaller than most countries. It is interesting to note, too, that in these countries sheep are rare and that the ox is pastured largely, and it has has been suggested that this animal probably acts as the common intermediate host for the disease.

At the same time it soon became recognized that the peculiar alveolar form was unknown in those countries such as Iceland, Algeria, Northern and Western Europe, South America and Australia, where the ordinary form was common. It is true, however, that a number of observers, having noted the frequency of peculiar multiloculated forms in the domestic animals of these countries, erroneously believed these to be identical with the alveolar form, and rather added to the confusion by denying the above statement. Dévé,[10] in a laudable attempt to overcome some of the confusion in nomenclature induced by the occurrence of so many morphological varieties both in man and animals, suggested that the name "*Echinococcus bavaro-tyrolienne*" should be given to the rare human form. The discovery, however, that the distribution of the disease was not rigidly confined to this area, led to the name not being generally accepted and Posselt's original designation, "*Echinococcus alveolaris*," is now universally used.

As interest in the subject grew, more and more cases were recorded, and it was soon found that, as many of the unicists had predicted, the above narrow geographical limitation did not exist. Cases were soon discovered and recorded in various parts of Russia by Romanoff,[11] Melnikoff,[2] Sabolotnoff[12] and others. Other cases were recorded in various parts of Germany and in the Jura region of France. No undoubted cases have as yet been recorded in Iceland, Algiers or Australia, nor, in spite of the great increase there in frequency of ordinary hydatid disease, in the republics of South America. It is true that, although Cranwell[13] maintained that the alveolar disease was unknown in Argentina, a case which has been verified histologically by Dévé was recorded there by Wernicke.[1] Unfortunately, no details have been forthcoming as to whether or not the patient was born in South America or was a European migrant, and the case must, therefore, be regarded as of doubtful authenticity.

It is not to be wondered that this peculiar distribution of the disease is still one of the most important arguments in favour of the dualistic doctrine of a separate tænia. The unicists have been compelled to attempt to explain why this rare disease is so common where the ordinary type is so rare and unknown where the ordinary type is so common. They have

replied, not very effectively it is true, with the suggestions: (i) That the disease has not the narrow distribution attributed to it, and that its range will in time be found to be wider. (ii) That in the particular countries where it is so common there exists some factor, soil, climate, racial characteristics of the people, even as Buhl suggested—a special hepatic tissue in the Tyrolese—that induces in some way or other the production of a parasitic variant. It has been suggested particularly that the effect of severe cold—these areas are mostly mountainous—on the ova is to kill off the greater number, the survivors escaping because they are endowed with greater resistance and so possibly greater virulence, with what Dévé has called *"une sorte de malignité désormais spécifique,"* so that when they do affect human beings, a more active type of lesion is produced.

PATHOLOGICAL CONSIDERATIONS.

A. Structural Characteristics of the Lesion.

Several active protagonists of the dualist doctrine have attempted from time to time to convince themselves and others that there are specific morphological characteristics which distinguish this parasitic lesion from all others. Melnikoff[2] in 1899 described a thin nucleated protoplasmic layer on the outer side of the hyalin layer, as well as on the inner side, and stated that this was the essential difference between the two forms. Posselt[14] in 1901 and 1906 strongly supported this view.

Dévé, however, using suitable staining methods, proved that this layer did not exist, and his work has been confirmed by Elenevsky[15] and Daujat.[16] There is no doubt that Posselt's observations were made on artefacts and none of the dualists now supports this view. It is an interesting commentary on the ardour with which this subject has been debated to note that Jenckel,[17] who was always a keen unicist, described, just as Melnikoff had in the alveolar form, an external budding of scolices in the typical hydatid cyst—a phenomenon which all authorities are unanimously agreed does not occur. It is needless to add that Jenckel used this erroneous observation as an argument in favour of the unity of the species.

When one considers the earliest developmental stage of the hydatid parasite, it is obvious that there cannot be an external protoplasmic layer. The nucleated germinal protoplasm—the "noble" element—is the only active part of the parasite and it elaborates not only the fluid, but also lays down the hyalin layer from within out as a protective layer to itself.

Although all are agreed that the mode of development of the scolices is essentially the same in all forms, yet many minor structural differences have been observed and a great deal of importance has been attached to these by some dualists, particularly Vogler, Mangold,[18] and Posselt.[14] On the other hand, these observations have been attacked by others both as regards their importance as well as their reality. The number of hooklets has been stated to be rather less on the average,

32 rather than 36 to 40. The size, too, as well as the shape of the scolical hooklets, is said to show some variation from the typical disease. Krabbe, Leuckart, Sabolotnoff and others have, however, shown that in ordinary hydatid disease the scolices and their hooklets show a very great range of variation, even in the same cyst, and that, therefore, these minor differences can be of little real significance. Most observers seem now to hold the same view.

In 1899 Melnikoff described in a case of alveolar hydatid disease special germinal elements that he regarded as embryonic forms. He believed that these had the power to migrate into the surrounding tissues of the host, there giving rise to a certain amount of cellular necrosis and a good deal of inflammatory reaction. He called these forms "*Jugendformen*" and laid great emphasis on their occurrence as a constant and typical feature of this particular lesion. He went, however, much further than this, and described a great number of so-called developmental forms. He believed that the parasite could multiply indefinitely in the human body, completing all stages of its life cycle here. There is no doubt that he was gravely misled, or, as Dévé rather succinctly puts it: "*L'auteur ne s'est peutêtre pas suffisamment tenu en garde contre son imagination.*"

It is strange, so strong is the influence of partisanship, that even as late 1912 a number of unicists accepted his description, but von Linstow, Jenckel, Dévé and even Posselt all attacked it within a few years, while in 1907 Elenevsky, one of the pupils of Melnikoff, in a long contribution to the subject, stated that he could not verify his teacher's findings and came to much the same conclusions as Dévé. The latter[4] pointed out that the fine granular protoplasmic masses described by Melnikoff were seminaked prolongations of the primary germinal membrane before this was covered with hyalin. They have the power of producing some toxin which has a necrotic action on the cells with which it comes in contact and which stimulates a marked cellular reaction on the part of the host. As he pointed out, the cuticularization of the germinal membrane in this parasitic form is irregular, delayed and may be for a long time deficient in certain areas, so that some part of the nucleated protoplasm may remain longer than others devoid of an outer protective layer.

B. Relationship with Hydatid Disease of Bone.

It is not surprising that, owing to the curious form assumed by the universally distributed hydatid of bone, its superficial resemblance to alveolar hydatid has been used by some writers in this controversy, and this has in consequence led to confusion. Because of the presence in it of innumerable small isolated cystic or semisolid hyalin masses set in a spongy matrix, often with a necrotic centre where the bone has been destroyed, and because of its slow persistent growth, Virchow[19] himself, made the first error by concluding that it was identical with the alveolar form first described by him. His authority in pathological matters

was so great that few dared to offer any criticism, and this view was not only accepted by most, but was at once used as an argument in favour of the unicist doctrine (Jenckel). Ganglophe,[20] too, in 1886 pointed out that practically all the accepted peculiarities of alveolar disease are present in bone hydatid disease and in a now classical thesis supported the unicists.

In 1895, however, Watson[21] first pointed out the now well-known fact that the same parasitic lesion, once it invades the periosseous tissues, alters its manner of growth. Under these conditions it becomes univesicular, lays down a thick laminated membrane, produces scolices and stimulates the tissues of the host to form a uniform adventitia, thus assuming all the features of a typical hydatid cyst. Wilms[22] in 1898 emphasized the same facts and drew attention to the differences between alveolar and typical osseous hydatid, stating that the peculiar form assumed by the latter was due solely to the nature of osseous tissue.

FIGURE VII. Section of hydatid of bone showing diverticula growing along bone canals. Note quiet pressure atrophy of bone and cellular reaction between bony trabeculæ. × 45.

Dévé[23] in 1905 also protested against the confusion that had arisen, and pointed out that only the grossest macroscopical analogies existed between the two lesions.

Cranwell,[24] from Argentina, also pointed out that there was no real connexion between the two lesions and emphasized the universal distribution of the osseous type.

In 1907 Elenevsky[15] reported a unique case in which a true alveolar hydatid of the liver had invaded the bone of the vertebral column, and observed that there the lesion did not resemble the classical osseous hydatid, but rather retained its characteristic alveolar features.

The writer[25] has come to conclusions similar to those of Dévé and Cranwell,[24] that is, that there is little resemblance between typical hydatid of bone and the alveolar form, and that the structure of the former is determined simply by the peculiar structure and resistant nature of osseous tissue. After lodgment of the embryo in the loose

medullary tissue a follicle forms, but owing to the confined space in the bony canals and their resistant walls, uniform spherical growth soon becomes impossible. The parasite grows in the line of least resistance along the bone canals, in the form of double layered prolongations extending centrifugally in all directions, producing by quiet pressure absorption and widening of the canals, a diverticulated sort of growth.

FIGURE VIII. Diverticulum from decalcified bone hydatid showing secondary closure and formation of small cyst. × 20.

Figure VII shows method of growth of the diverticula along the bony canals.

Localized swellings appear in these diverticula in which the nucleated protoplasm may form fluid and lay down fresh laminated membrane, so that small cysts which become secondarily cut off are produced, the remains of the connecting hyalin strands being removed by phagocytosis (see Figure VIII).

At times, when the space becomes large enough or when the comparatively large medullary cavity of the bone is entered, a relatively large cyst one centimetre to two centimetres in diameter, which may also be fertile, may be formed. Once the periosteum is eroded and the soft

tissues are invaded, the whole pathological picture alters, the parasite forming typical, often fertile, unilocular cysts.

The only value in this controversy which the numerous observations on bone hydatid have had, is to emphasize in support of the unicist doctrine the fact that hydatid disease in man can often assume peculiar forms, and that very often the nature of the surrounding tissues may exert a dominant influence on the structure of the resulting parasitic growth. These two growths have so little in common that it is difficult, however, to allot much weight to the arguments of Wilms and Jenckel, both ardent unicists, who see in osseous hydatid a marked tendency towards a transitional form between alveolar and typical hydatid.

C. Special Features of the Alveolar Form.

Dévé, who has always been a supporter of the dualist doctrine, lays importance on some special characteristics of the alveolar disease. He points out that apart from the obvious differences in structure, transitions between this and the common form have not been recorded to his knowlege; that the characteristics of the lesion remain constant in all tissues and in the case of metastases, and that its persistence, its toxic influence on the surrounding tissues and its relentless peripheral extension give it a certain degree of malignancy.

After discussing the characteristics at length and assessing their value, he is forced to admit, however: (i) That after all the structure of alveolar hydatid presents the same fundamental features as ordinary hydatid disease, that is, the same sort of laminated membrane, a similar inner nucleated layer, which functions in the same way as in the typical disease in the production of fluid, laminated membrane and reproductive elements. (ii) The only essential difference lies in the great activity of the germinal protoplasm with its power of persistent irregular uncoordinated growth and of producing intense toxic and necrotic changes in the tissues of the host.

Nevertheless, these peculiarities are distinct and constitute a forceful argument in favour of the dualist theory. The unicists maintain, however, that the difference is one of degree only, that it is not so marked as Dévé and others maintain, and, further, that, just as in the case of *Cysticercus racemosis* and *Cysticercus celulosæ*, which are very dissimilar larval forms of the same parasite, it can be explained by parasitic variations in which there is some dissociation of the coordinated and balanced growth of the parasite—similar views to those advanced by the writer.[8]

Complement Fixation Reaction.

The use and value of the detection of specific complement fixing properties in the serum of patients harbouring hydatid disease are now well established, and it might be expected that some information of value in respect to this question would be forthcoming from a study of this reaction. In 1910 Dobrotine[26] first obtained a positive fixation

reaction in a case of alveolar disease, using ordinary hydatid fluid as an antigen. Skaczewsky[27] and Mollard and Favre[28] in 1911 have also recorded positive findings, while in the case I have here recorded strongly positive fixation occurred. Unfortunately, we know that among the helminths cross-reactions are relatively common, even in groups as far apart as *Echinococcus granulosus* and *Tænia saginata*, so that the fact that the common antigen is efficacious cannot be an argument in favour of the unity of the species.

RELATIONSHIP TO HYDATID DISEASE IN ANIMALS AND THE QUESTION OF THE LIFE CYCLE.

Many observations have been made on the pathology of hydatid lesions in the various domestic animals, contributions having been made by workers in many countries. These animals act as intermediate hosts in the same way as man, are undoubtedly infested through the common medium of the dog, and in all countries their lesions show the same series of surprising morphological variations. All are agreed that the most interesting and characteristic lesions occur in the liver of the ox, the lungs in this animal being often affected with much less atypical cystic forms. This variation in the form assumed by the larval stage of what must be, when the ætiological possibilities are considered, a common tænia, in the same or different organs of the same animal very often, is a very interesting and important fact. In order to obviate confusion, the rather complicated multicystic lesions, which are very often sterile, have been designated *Echinococcus multilocularis*.

All degrees of complexity from simple univesicular to complex lesions in which there is a strange mixture of parasitic and fibrotic adventitious tissues, exist both in the ox and sheep, although in the latter animal complex forms are less common. The fact that these complex lesions sometimes have an alveolar appearance, that they show, at least on superficial examination, many resemblances to *Echinococcus alveolaris*, has naturally led to erroneous deductions as to the frequency of the latter and has also been used freely in this controversy by protagonists of both sides.

The earlier observers pointed out the general resemblances between the two lesions, but emphasized the absence of central necrosis, due, according to Railliet and Morot,[29] to the relative youth of the slaughtered animals. Some of them frankly admitted that they could not decide whether or not the classical *multilocularis* of the ox was distinct from the alveolar form. Dévé, however, maintained that the two lesions presented many essential differences. He pointed out that in the bovine form the cysts were relatively large and regular, sometimes being full of fluid, the laminated membrane separating easily, that central cavitation was unknown, that transitions to the hydatid form were quite common in the same lesion and that the lesion tended to be circumscribed, having no trace of the malignancy of the alveolar form. The lesion, as seen in the ox, tends to grow by an external

budding of diverticula, with secondary exclusion of separate cysts, thus giving rise to congeries of cysts set in a fibrous matrix. This has been pointed out by Prougeansky[30] and the writer[8] (see Figure IX).

The cellular reaction of the host tissues is also regular and well organized, consisting usually of a trizonal arrangement of cells, the inner cells being composed of eosinophile and leucocytic cells, the middle of radially arranged endothelial cells and the outer of fibroblastic cells. This very constant finding in all bovine cysts is evidence, I think, of

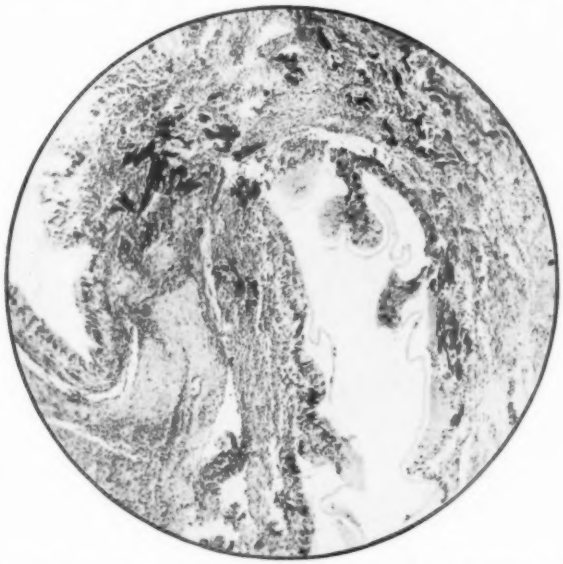

FIGURE IX. Section of *Echinococcus multilocularis* of the liver of an ox showing progressive diverticulation, poor type of hyalin, and trizonal arrangement of the cellular reaction. × 60.

the persistent struggle of the host against the parasite and supports the statement put forward by the writer that the ox is by no means an ideal intermediate host. It is true that Dévé himself has rather modified his former view in that he states, after discussing his belief as to the distinction that must be made between *Echinococcus alveolaris* and *Echinococcus multilocularis* in the ox and sheep: "*Nous n'oserions plus l'affirmer aujourd'hui. Il est possible en effet que la même lésion ait changé de caractère avec le terrain.*"

The South American observers, Llambaïs[31] and Parodi,[32] have made some interesting observations on the early stages of development of *Echinococcus multilocularis* in the ox. They state that sometimes in this form of the disease peculiar granular "embryonic" buds can be seen growing outside the confines of the original cyst and extending

freely into the tissue of the host. These they regard as similar to the appearances described in the alveolar lesion, that they represent transitional forms between the two lesions and on this ground they support the unicist theory.

The writer[8] has pointed out that purely histological methods can scarcely carry us further in this problem, that parasitological variation is common in other diseases, and that it is to be expected that transitional forms between the various hydatid lesions should occur. The occurrence, in the same or different organs of the same animal such as the ox, of lesions which must be produced by the same parasite and which are morphologically so dissimilar, is proof of this. The more experience one has with this disease in man, too, the more one is struck by its remarkable larval pleomorphism.

The exact relationship between the universally described *Echinococcus multilocularis* of animals and the rare human alveolar form is also of fundamental importance because of the question of the life cycle of the latter disease. It is obviously essential that it must have another host besides man, otherwise how, if, as the dualists in general maintain, the alveolar form is confined to man, can the life cycle be completed?

Some of the dualists have stated that the wolf may be the definitive host, but wolves are unknown in some of the endemic areas; others have postulated a specific breed of dog. Klebs and Posselt believed that the alveolar form could occur in the ox and that dogs are infected by eating the liver of this animal. It would seem, therefore, that the dualists are faced with an unanswerable question as to how the life cycle is completed, unless they believe that the ox acts as the intermediate host of this special parasite, and that, therefore, the *Echinococcus multilocularis* is the alveolar form in the ox and so has a world distribution.

ANIMAL EXPERIMENTATION.

It would be thought at first sight that the whole question of a specific tænia could be solved satisfactorily and completely by feeding dogs with alveolar hydatid lesions, obtaining the tænia and reproducing the larval form by feeding appropriate animals. The problem, however, is not so simple as it looks; really it bristles with difficulties, mainly of a technical nature. In the first place, the disease is relatively rare. It is rarely diagnosed clinically; indeed it is often an operative or *post mortem* surprise. As a result, properly controlled young dogs are rarely ready. Also these lesions are often sterile and the number of scolices is never great, so that the dog may either not become infested or the infestation would be so slight that it would be difficult to detect the tænia or to recover enough fresh ova for feeding experiments. If the dog did become infested, some three or four months must be allowed to lapse for the tæniæ to reach maturity before an attempt is made to infest an intermediate host. These hosts, too, are not so easy to infest as one

would expect and following the attempt some months must elapse before the cysts, if present, could with certainty be identified.

In 1883, five to nine weeks after feeding a dog two years old with fragments of an alveolar lesion, Klemm[33] discovered a great number of mature tæniæ, which he regarded as identical with the *Tænia echinococcus* of Siebold. The obvious objection that the dog used was not verified free from all tænia beforehand was at once raised. It is also very unlikely that feeding even with a very large quantity of this notoriously unfertile lesion could give rise to as many worms as he described.

Under similar conditions Mangold[34] in 1892 obtained three tæniæ which in his opinion showed specific characteristics. He fed a sucking-pig with the intestines of the dog a day after its death, and in the liver of the pig, killed four months later, he found two small alveolar lesions about as big as a walnut. Dévé criticizes this experiment, because the exposure of the tæniæ to putrefaction for a day at least would be very apt in any feeding experiment to lead to negative results. Also, the alleged resultant lesions in the pig were much too large to have been produced in a mere four months, because even at five months ordinary experimental hydatid cysts in these animals are never as large as this. In the absence of all controls this experiment is generally regarded as inconclusive.

Posselt[34] in 1902 conducted the first series of experiments under rigorous scientific conditions and, after feeding young dogs, obtained specimens of a tænia which he regarded as different from the common *Tænia echinococcus* of Siebold. He laid great stress on the number, the shape and size of the hooklets and on the arrangement of the ova in the terminal segment. He was so sure of the significance of these observations that he described this tænia as a new species, *Tænia echinococcus alveolaris*.

Jenckel[35] protested strongly that, as had been found out years before by Leuckart, von Linstow and others, the common *Tænia echinococcus* exhibited all these variations and that, therefore, Posselt's action in naming a new species was absolutely unwarranted. Posselt, in spite of careful experimentation, was unable to produce the larval stage of the tænia he regarded as a distinct form in any animal, so that his experiments must be regarded as inconclusive. Other attempts to repeat the full experiment have been made, but nobody has yet obtained positive results. As Dévé points out, no facts of unimpeachable value to support either doctrine have as yet emerged from the experimental methods, but there is no doubt that carefully conceived and fully completed feeding experiments will indisputably solve the problem. He has laid down very fully all the factors which must be fulfilled in the conduct of such experiments.

DISCUSSION AND SIGNIFICANCE OF PRESENT CASE.

From the study of the evidence on which the two doctrines are based we find that the dualists put forward two very important argu-

ments. They are: (i) the comparatively narrow geographical distribution, (ii) the peculiar pathological appearance of the lesion.

Neither of these has as yet been completely answered by the unicists, who in their turn counter with the very pertinent and at present absolutely unanswerable question: If the lesion is a specific one, confined to man, how can the life cycle be completed by passage through a definitive host and the parasite prevented from becoming extinct?

The Geographical Distribution.

We have seen how in course of time it has been discovered that this lesion has a wider geographical range than once thought. There is no doubt that several foci exist in Russia, that it has been found in France and Germany, that a possible case has been recorded in Argentina, and now the first case has appeared in Australia in a person who has never left that country. It seems, therefore, that the geographical argument has lost all its value and that it must be for ever dropped by the holders of the dualist doctrine. The writer believes that in the future more careful records will reveal sporadic cases in all countries and that the discovery of the case here recorded is an argument in favour of the essential unity of the hydatid species.

The Pathological Appearances of the Lesion.

Great importance has been attached to these, particularly to the persistent infiltration of the host tissues, the comparative malignancy, as it were, of the process as compared to the common hydatid disease, due, as Dévé maintains, to a special primitive biological property of the nucleated protoplasm of the parasite which seems to be endowed with special activity as regards growth, and to the fact that obvious transitional forms between it and other forms of hydatid disease do not occur. Dévé and other dualists are prepared to admit, however, that in real essentials all the lesions have much in common, and that the difference between them is a matter of degree only.

In spite of the fact that the lesion here reported fulfils all the conditions which are laid down as requisite for alveolar hydatid disease, it presents some unusual features.

In the first place, I believe that the lesion in this case is extremely old and of very slow growth, showing persistence rather than malignancy or activity. It is possible that the patient was, like the majority of those harbouring hydatid disease, infested while young, and that the lesion has been present practically since infancy. It is difficult from the other recorded cases to gather whether there is any evidence in them to support this view. The size of the lesion, the compensatory hypertrophy of the left lobe of the liver, the density of the sclerotic tissue on the right side and under the diaphragm, where it measured eight millimetres (one-third of an inch) in thickness, the amount of fibrosis, the degree of calcareous change in the upper part of the adventitia, and the relatively quiet nature of the reaction at the periphery, support the view that the condition had been present for many years.

It is possible, then, that the parasitic infiltration is really only an index of parasitic persistence at a very low level of vitality. This is also indicated by the amount of parasitic degeneration and the lack of reproductive elements. The central necrotic change has been, of course, induced by gradual interference with the blood supply, by dense fibrosis to which was added a mild degree of local toxæmia. This cavitation and the rupture into the gall bladder should, I believe, be regarded as the terminal event of a long standing pathological process occurring in a man of sixty-three years.

In the second place the lesion shows the following differences from classical alveolar disease:

1. The cavities, filled with folded, non-adherent, laminated membrane which can be shelled out of many with ease, are relatively larger than those usually seen, several of them being about two centimetres in their largest diameter. In addition, where the process was extending, the surrounding hepatic tissue showed a systematic organized attempt to combat the parasitic growth. The cellular reaction here assumed a trizonal arrangement very like that seen in typical *Echinococcus multilocularis* (compare Figures VIA, VIB, VIC, VID and IX).

2. The appearances in the subperitoneal zone and in the region of the portal fissure of seven small single cysts with definite adventitiæ, which resembled in all particulars small hydatid cysts, except that they were not fertile. These showed no tendency towards infiltration.

3. The respect that the whole lesion had for the peritoneum and the diaphragm. While infiltrating the liver tissue widely, it made no attempt to invade those tissues which had elaborated a sclerotic adventitia. In the same way, although the lumen of the *vena cava* was somewhat narrowed by the lesion, there was no suggestion of any definite invasion of this or any other vein.

4. There was no suggestion, in spite of the extent of the lesion, of any involvement of lymphatic glands, nor were there any blood-borne metastases in other organs.

5. The presence in the large central cavity of three small cysts which somewhat resembled daughter cysts. These were thin walled and sterile, and it is probable that they were extruded from the seminecrotic trabeculated lining of the cavity following the trauma of exploration. It is interesting to note that in a case of proved alveolar hydatid of the brain von Hibler encountered a somewhat similar phenomenon. As noted above, it is very doubtful, however, whether they can be regarded as analogous to the common endogenous daughter cysts so commonly seen in ordinary hydatid disease.

This lesion, I believe, exhibits features which are common to both *alveolaris* and *Echinococcus multilocularis*, bearing out the writer's contention that transitional forms between all the various morphological variants of hydatid disease can occur, and that there is no need to postulate the existence of two specific tæniæ.

CONCLUSIONS.

A consideration of this lesion has confirmed the writer in his opinion that all the hydatid lesions that have been described, either in man or animal, are essentially the same and that the pleomorphism they exhibit is due to the occurrence of parasitic variations. In all these cystic forms there is an active nucleated protoplasm, which elaborates fluid and laminated membrane and, if conditions are favourable, reproductive elements in the form of scolices. Normally these functions are coordinated to insure parasitic survival and perpetuation of the species, but under certain, at present unknown, conditions there occur variations from the normal growth, such as may happen in any parasitic lesion. This is probably influenced partly by the *terrain*, a factor which is too often not considered at all in discussing the problem. How else can we explain the morphological variations seen in the ordinary hydatid cysts of man or the herbivora? It would seem that when this variation occurs, a dissociation of the growth regulation takes place, one or other of the larval developmental phases gaining the ascendency. If the parasite retains its early growth activity, it grows steadily, elaborates a diffusible toxin which causes cellular necrosis and invades the host tissues, without attempting to form fluid to lay down a limiting hyalin layer or to give rise to scolices. Under these conditions a type of growth which resembles the alveolar form in all essentials will be produced. It is possible, as indicated above, that a deficiency in the powers of the parasite to lay down hyalin might be the determining factor in inducing this type of growth and that once some degree of cuticularization occurred, the parasite would be able to retain fluid and produce scolices. At the same time, an adjustment of its relationship with the cells of the host would be arrived at and an attempt made by the latter towards encapsulation of the older parts of the lesion. If the next phase of development were reached, in which the cellular reaction of the host was well organized and persistent, the lesion would assume the form so commonly seen in the ox. Here, too, the hyalin layer is also somewhat deficient, although not in the same degree as in the last type. The cells of the host are not overcome, but react vigorously with the production of a trizonal cellular layer, which effectually resists parasitic growth. It is possible that in this type there is just sufficient leakage of parasitic toxin to stimulate this reaction on the part of the host tissues, or it may be that we have here an example of the peculiar excessive cellular reaction of bovine tissue to all forms of chronic irritation. There seems to be little doubt that in *Echinococcus multilocularis* of the ox the parasite is usually just managing to survive. This is exemplified by the frequency of degeneration, the frequent absence of fluid containing cysts, the frequent sterility of the lesion, the active phagocytosis of parasitic remnants and the great amount of fibrous tissues, which limits parasitic extension in this animal. If the third phase of development is reached, a perfect symbiotic balance is struck between the parasite and the host, the functions of the nucleated protoplasm of the parasite being accurately

coordinated. After establishing itself, this elaborates an efficient protective hyalin laminated layer, which retains the specific fluid, prevents its leakage from stimulating an aggressive action on the part of the host cellular defences, and allows the maintenance of ideal conditions for the development of scolices.

If these conditions are fulfilled, typical, fertile, univesicular, well encapsulated hydatid cysts so commonly seen in man or the sheep are produced.

The explanation of the cause of this parasitic variation can only be conjectured. I should like to make, however, the tentative suggestion that it may be due to passage of the larval stage through the bovine liver. The typical multilocular lesion of the ox is notoriously sterile and in Australia at least, having regard to the method of slaughtering and the comparatively low incidence of hydatid infestation in this animal, I do not believe that dogs are very often infested from this source. There can be no doubt also that as, practically speaking, none of our herbivorous animals originally came from any of the countries where alveolar disease is endemic, and as the lesions in all our animals show the same morphological variations and the same transitions as animals in all other countries, in them at least, the common source of infestation is the dog, who is practically always infested from the sheep, the ideal intermediate host. It is possible, however, that occasionally a dog is infected from ingesting the liver of an ox and might, in turn, infect a human being. This would obviously be a very rare occurrence in a country such as Australia with so scattered a population and with its peculiar ætiological factors. In those countries where the alveolar disease is common, there seems little doubt that the total number of human cases of both types of hydatid disease is, in spite of their being fairly thickly populated by a peasant class, relatively small.

In these countries, too, in marked contrast to those where the ordinary disease is usual, the commonest domestic animal is the ox. The fact that the lesions in the ox show considerable resemblances to the alveolar form, particularly in the way of deficient hyalin production and quiet, persistent, irregular growth, suggests that such a larval lesion might be able to transmit these characteristics. Is it not possible, as Posselt, though he was a dualist, originally believed, that these people may be infested through the medium of this intermediate host? This would explain the relative infrequency of hydatid disease, the proportionately greater occurrence of the alveolar form among them, and obviate the difficulty of explaining how the life cycle was completed. It would seem, therefore, that if experimental methods remain inconclusive, a careful ætiological study of the possible sources of infestation of the dog in those countries where the disease is endemic, might give important information.

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REFERENCES.

(1) R. Wernicke: Quoted by F. Dévé, Reference Number (3), page 379.

(2) R. Melnikoff: *"Ziegler's Beiträge zur Pathologischen Anatomie und Physiologie,"* Supplement, 1901.

(3) F. Dévé: *"Échinococcose Alvéolaire et Échinococcose Hydatique," Premier Congrès International de Pathologie Comparée*, Paris, October, 1912, page 363.

(4) R. Virchow: *"Die multiloculare ulcerirende Echinokokkengeschwulst der Leber," Wurzburgen Medizinischen Gesellschaft*, March 10, 1856, Volume VI, page 84.

(5) F. Küchenmeister: *"Die Parasiten des Menschen,"* 1879, page 162. Quoted by F. Dévé, Reference Number (3), page 364.

(6) R. Leuckart: *"Die Parasiten des Menschen,"* 1879.

(7) F. Morin: *"Deux Cas de Tumeur a Échinococcose alvéolaire du Foie," Thèse de Berne*, 1875. Quoted by F. Dévé, Reference Number (3), page 365.

(8) H. R. Dew: "Observations on Hydatid Disease in the Domestic Herbivora (Echinococcus Multilocularis) and its Relationship with Echinococcus Alveolaris," *The Medical Journal of Australia*, September 4, 1926, page 301.

(9) W. Schmidt: *"Ueber die geographische Verbreitung des Echinococcus multilocularis und hydatidosus in Bayern auf Grund der Münchner Fälle,"* Inaugural Discussion, München, 1809. Quoted by F. Dévé, Reference Number (3).

(10) F. Dévé: *"Sur quelques caractères zoologiques de l'échinococcose alvéolaire bavaro-tyrolienne," Comptes Rendus de la Société de Biologie*, Paris, January 21, 1905.

(11) F. Romanoff: *"Zur Frage des vielnestigen Echinococcus," Aus der Universität zu Tomsk*, 1892. Quoted by A. Posselt, Reference Number (14).

(12) Sabolotnoff: *"Noch einige Bemerkungen über multilocularen Echinococcus," Tageblatt der Aerztegesellschaft an der Universität Kasan*, December 4, 1898. Quoted by A. Posselt, Reference Number (14).

(13) D. J. Cranwell: *"Kystes hydatiques de l'humerus," Revue d'Orthopédie*, November 1, 1907, page 513.

(14) A. Posselt: *"Die Stellung des Alveolarechinokokkus," Münchener Medizinische Wochenschrift*, Number 12, 1906, page 537.

(15) K. Elenevsky: *"Zur pathologischen Anatomie des multilocularen Echinococcus beim Menschen," Archiv für klinische Chirurgie*, Volume LXXXII, 1907, page 393.

(16) Charles Daujat: *"Échinococcose alvéolaire principalement du foie. Étude critique a propos d'un cas français," Thèse de Lyon*, 1912. Quoted by F. Dévé, Reference Number (3).

(17) A. Jenckel: *"Beiträge zur Pathologie des Alveolarechinococcus," Deutsche Zeitschrift für Chiurgie*, Volume LXXXVII, 1907, page 1.

(18) C. Mangold: *"Ueber den multilocularen Echinococcus und seine Tanie," Berlin Klinische Wochenschrift*, Number 3, 1892, page 50.

(19) R. Virchow: *"Knochen Echinococcus," Virchow's Archiv*, 1880, page 183.

(20) M. Ganglophe: *"Kystes hydatiques des os," Archives générales de Médecine*, Volume II, 1886, page 345.

(21) A. Watson: "Hydatid Cysts of Bone," *Australasian Medical Gazette*, April, 1895, page 161.

(22) R. Wilms: *"Echinococcus multilocularis der Wirbelsäule und das Verhaltniss des multilokularen Echinococcus zum Echinococcus hydatidosus," Beiträge zur Klinische Chirurgie*, Volume XXI, 1898, page 151.

[23] F. Dévé: *"Échinococcose alvéolaire humaine et échinococcose multiloculaire bovine,"* *Anales de la Facultad de Medicina de Monte Video*, March, 1920, page 129.

[24] D. Cranwell: Quoted by Dévé, Reference Number [3].

[25] H. R. Dew: "Hydatid Disease," 1928, page 370.

[26] A. N. Dobrotine: *"Contribution au diagnostic de l'échinocoque multiloculaire a l'aide de la réaction biologique de la déviation du complement,"* *Roussky Vratch*, Volume IX, 1910; abstracted in *Journal de Chirurgie*, Volume V, 1910, page 320.

[27] G. Skaczewsky: *"La signification clinique de la réaction de Weinberg pour le diagnostic de l'échinocoque,"* *Roussky Vratch*, Number 17, 1911; abstracted in *Münchener Medizinsche Wochenschrift*, November 28, 1911.

[28] Mollard and Favre: *"Échinococcose alvéolaire du foie,"* *Société médicale des Hopitaux de Lyon*, November 14, 1911; *Lyon médical*, November 26, 1911.

[29] H. Railliet and F. Morot: *"L'échinocoque multiloculaire observé en France chez les animaux,"* *Bulletin de l'Académie de Médecine*, Volume XXXIII, 1898, page 428.

[30] M. Prougeansky: *"Ueber die multilokulare ulcerierende Echinococcusgeschwulst in der Leber,"* Inaugural Discussion, Zurich, 1875. Quoted by F. Dévé, Reference Number [3].

[31] J. Llambias: *"Equinococo alveolar de los bovinos,"* *Anales de la Facultad de Medicina de Monte Video*, 1919, Volume IV, page 585.

[32] S. Parodi: *"Unidad o Dualidad de la Tænia Echinococcus,"* *Segundo Congreso de Medicina*, Buenos Aires, October, 1922, page 44.

[33] A. Klemm: *"Zur Kenntniss des Echinococcus multilocularis der Leber,"* Inaugural Dissertation, München, 1883. Quoted by F. Dévé, Reference Number [3].

[34] A. Posselt: *"Die Stellung des Alveolarechinokokkus,"* *Münchener Medizinsche Wochenschrift*, 1906, Volume LIII, page 537.

[35] Jenckel: *"Beitrag zur Chirurgie der Leber,"* *Deutsche Zeitschrift für Chirurgie*, Volume XCVI, 1908, page 338.

NATURE AND ART IN THE HEALING OF FRACTURES.

By Ernest W. Hey Groves,
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It would seem almost impossible to say anything new about the treatment of fractures of the long bones. But it is worth while to review some of the modern methods, especially in regard to traction appliances, the rôle of direct operation and the treatment of infected open fractures. In several respects the problem is much more difficult today than it was even a generation ago. Then manipulation and splinting were the only available methods for closed fractures, whilst a badly infected open fracture was treated by amputation. But now the X rays have made early and exact diagnosis easy, and at the same time have greatly increased the possibilities of exact reposition and have widened the range of treatment. Direct open operations of all kinds have been perfected, and this too means that the choice of treatment is correspondingly greater. And in dealing with infected open fractures, the resources of antiseptic surgery have made possible the saving of limbs which used to be sacrificed. But all the resources placed at our disposal by the X rays, modern operative technique and aseptic surgery, make it incumbent upon us to examine and understand the natural processes of the healing of bone injuries much more exactly than in those days when conservative and expectant treatment represented the routine methods. It is essential that we should understand the natural process of repair, so that we may aid and not hinder vital growth. My meaning may perhaps be made more clear by an illustration from the analogous problem of the healing of any open cutaneous wound such as that made by the operation for the radical cure of an inguinal hernia.

The actual healing of such a wound is a natural process for which there can be no artificial substitute, but, on the other hand, correct surgical art can so work with Nature as to make natural healing rapid and perfect, whilst bad technique will make it slow and imperfect. Asepsis, hæmostasis, accurate stitching, an absence of tension, sharp needles and proper suture material are some of the factors in the artificial closure of the wound which will aid and simplify the natural process of repair; whereas sepsis, oozing of blood, masses of tissue caught up in tight stitches which turn the edges in are the factors which will delay repair and usually will lead to loss of tissue by suppuration or sloughing, with final healing by granulation. In the repair of fractures

the essential principles are the same, but they are rendered rather more complicated by the peculiar vascular conditions of the bone and the great difficulty in attaining and maintaining correct opposition and complete rest in the injured limb.

THE NATURAL HEALING OF FRACTURES.

The ordinary text book description of the healing of a fracture with the orderly succession of blood clot round the fragments, granulation tissue, callus, rarefaction of the bone ends with ossification of the callus and final union by a process of incorporation of ossified callus with partially deossified bone, is applicable only to fractures in which there has been little or no displacement. If we study a case in which Nature has been left unaided, we soon realize that simple and ideal healing is impossible. Either mal-union or non-union will take place. If the displaced bone ends are not deprived of their blood supply, they will produce callus in a large quantity, but the callus mass at each end will have difficulty in establishing relation with that of its fellow and even when the gap has been bridged the constant movement of the limb will make it most difficult for fusion of the callus masses. In fact, it is only if sufficient callus is thrown out to form a crude putty-like internal splinting that union will occur, and this naturally will be gross mal-union, with callus excess.

On the other hand, the bone ends, especially the hard bones of the adult skeleton, when violently broken and torn asunder, frequently become devascularized and for some time literally constitute a form of aseptic necrosis. Such avascular bone ends present a most formidable hindrance to repair. If the bones are replaced in good position and kept at rest, the dead ends may in time become invaded by vascular and cellular tissue which penetrates its empty canals in much the same way as occurs in the vascularization of a transplanted bone graft. But if any unfavourable factor is introduced, such as movement of the parts upon one another, the introduction of foreign bodies or any sepsis, then the bone ends remain to all intents and purposes dead and inert. Non-union with the formation of a false joint occurs, with a sclerosis or eburnation of the ends of the bone.

This liability to devascularization, indolence and aseptic necrosis of the bone ends in a fracture is a factor of very great importance which is not sufficiently recognized in treatment or prognosis. It means that in every fracture with displacement there is a very critical phase in which circulation and vitality of the bones are just in the balance, so that a very little help in the right direction will restore the feeble tide of life, whilst a very little injudicious action will destroy it. These points are well shown in the experiments here illustrated. They prove that when a fracture is complete, mal-union or non-union is the natural result, and that aseptic necrosis of the bone ends plays an important part in producing the latter result. There are many clinical evidences, too, of the fact that a poor blood circulation in the bone ends is liable

to cause delayed union or non-union. Of these, the most notable are the fracture of the neck of the femur and that of the lower third of the tibia. In a fractured femoral neck the upper fragment has a very feeble blood supply through the *ligamentum teres* and the untorn reflected fibres of the capsule. Therefore, unless accurate apposition with the vascular distal fragment is made at once and maintained efficiently, non-union will occur. In the tibia, broken below the middle, the lower fragment is robbed of its blood supply from the nutrient artery and the dense bone offers great hindrance to the process of revascularization. If then it is admitted that the natural healing of a displaced fracture has a great tendency to mal-union or non-union, it is necessary to inquire what effects artificial interference will have in preventing or increasing this natural tendency.

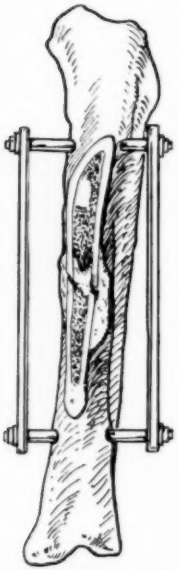

FIGURE I. Cat's tibia fourteen days after fracture. Treated by double transfixation and showing ideal early repair.

It is well to begin with experimental evidence. This demonstrates quite clearly the paramount importance of three things, namely, (i) accurate replacement of the broken bone ends, (ii) firm fixation of the bone during the early stages of repair, (iii) the avoidance of direct interference with the ends of the bone by operative manipulation and the use of foreign bodies.

In animal experiments I have found that accurate placing of the bone ends and firm fixation can be most effectively carried out by a double transfixation apparatus shown in Figure I. It may be fairly claimed that with this appliance the most ideal healing is produced. That is to say, there is just enough callus formed and this is placed at just the right place, whilst within a period of six weeks the bone is restored almost exactly to its original shape and strength. In marked contrast to this perfection of result is the effect of using metallic plates, screws, pins or wire. Short plates and screws always become displaced and the fractured ends fall apart. Usually the part of bone from which the plate has been dragged away shows necrosis. It is impossible to doubt that in such a condition the plating has actually made ultimate union more difficult. This is the state of affairs which generally exists in the not infrequent clinical cases in which obstinate non-union follows a plating operation. If, however, a really massive plate is used so that the real fixation of the bone is made by holding it by parts at a distance from the actual break, then union and good function will follow. But the actual healing process at the seat of fracture is slow and imperfect, whilst the bone as a whole becomes the seat of an active proliferative inflammation, which cannot be regarded as normal or desirable and which in human beings is liable to be followed by late symptoms of pain or sinous formation.

THE ADVANTAGES OF USING LIVING MATERIAL RATHER THAN FOREIGN BODIES IN THE DIRECT FIXATION OF FRACTURE.

Before proceeding to develop my main argument in favour of the indirect fixation of fractures by transfixation and traction methods, I should like to dwell for a moment on this important point of the great advantage of using living material rather than metal substance for the direct fixation of fractures. I can do this best by two examples.

The first is from a soldier, during the war. He had had a fracture of the left humerus, which had failed to unite and had therefore been operated upon before he came under my care. When I saw him three months later, his arm was very painful and there was distinct mobility with grating at the site of fracture. An X ray examination made the cause of this condition quite plain. The fracture had been fixed by a short plate and five screws; evidently even at the time the fixation seemed insecure, and the operator used several turns of wire round the bone to fix it to the plate. But this conglomeration of foreign bodies round the fracture caused merely an outpouring of connective tissue, which encapsulated the part, but there was no callus formed by the bones, the circulation in which had been so seriously interfered with. There had been, however, no suppuration and I would emphasize this point, because in my experience the failure of plating has frequently nothing to do with infection.

I operated upon him and removed the plate, screws, wire and a quantity of dense fibrous tissue deeply stained with metallic oxide. The bone ends were refreshed and united over an intramedullary bone graft taken from the tibia. The bone united quite smoothly, and it is clear that the bone which stubbornly refused to throw out any reparative material when surrounded by foreign metal, responded by natural healing when held by natural bony tissue.

The other example is of the fractured patella. As far as immediate results are concerned nothing could be more satisfactory than the treatment of a transverse fracture of the patella by encirclement with a stout wire. But after the lapse of time in many cases there is an undesirable development. The bone proliferates round the wire and a condition closely simulating osteoarthritis is produced. In some cases the wire becomes broken and its fragments wander into the tissues.

Thus in the case figured a woman had the patella united by wire fifteen years ago and had had a rapid and perfect recovery. Of late years she had developed some pain and stiffness in the knee and the skiagram showed that the wire had broken and that one part of it was lying loose in the knee joint (see Figures IIA and IIB).

It is true that by using iron instead of silver wire this tendency to disintegration of the metal suture may be prevented, but the proliferative reaction of the bone still remains and will cause definite pain and grating. Contrast with this the smooth and natural healing after the use of fascial sutures. In dealing with the patella the best possible natural suture material lies close at hand, the strong ilio-tibial band of the *fascia lata* being actually adjacent. A strip of this fascia is freed 15 centimetres (six inches) above the knee, whilst its lower attachment to the tibia is retained. This is then wound round the patella, the quadriceps attachment being pierced above and the patella ligament below. Used in this way, it not only makes a perfect suture, but it affords a direct attachment of the quadriceps tendon and upper patellar fragment to the tibia, independent of that of the natural patellar ligament.

TRACTION METHODS.

All clinical and experimental evidence goes to show that axial traction is the best method for the treatment of fractures of the long bones which present marked displacement. It restores the limb to its normal length and causes the broken fragments to fall together, whilst the vitality and vascular supply are not endangered. But though it is universally agreed that pulling in the long axis of the bone is an

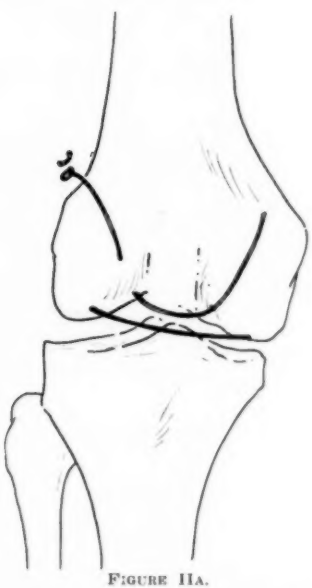

FIGURE IIA.

FIGURE IIB.

Outline tracing of skiagram of fracture of the patella which had been treated by silver wire circulage fifteen years previously. Note the arthritic changes in the patella and the way in which one fragment of the wire has wandered into the joint.

essential part of the treatment, very wide differences of opinion exist as to the method of applying this force and of maintaining it. Should the traction force be applied for the moment and the bones then fixed, or should the pulling be allowed to act over a long time? Should the traction be applied indirectly by adhesive appliances on the skin or directly by tongs or pins applied directly to the bone? I do not think that any of these questions can be answered dogmatically in a general sense.

The treatment of fractures is a matter in which judgement must be guided by experience and every case treated on its own merits. It would be sheer waste of time if I made any attempt to describe the various mechanical devices which have been introduced in recent years to accom-

plish the axial traction of fractures. They vary in complexity from a simple stocking stuck on to the leg by a special glue to a complex scaffolding surmounting the bed and provided with innumerable pulleys and weights, reminding one of the elaborate fooling pictured in Heath Robinson's cartoons. I will confine myself to describing three methods of traction, each of which has its special indication and advantages.

Wire Traction.

Of late years, and chiefly in Germany and Switzerland, skeletal traction has been carried out by means of a fine steel wire used as a transfixion pin, and this taut wire traction has been exalted into a new method and elaborated by many additional devices. It will be convenient to describe its method of application first before discussing its advantages and drawbacks. If applied to a broken femur, the following is the technique for putting on Kirschner's pattern of the taut wire. The wire used is made of hard rustless steel, supplied in straight pieces twenty centimetres long and three millimetres thick, each piece being pointed at one end. One of these steel wires is fixed in a holding chuck which in its turn is fitted to an electric motor, for example, that which drives Albee's saw. In order to steady the long rather flexible wire drill, it is held in a collapsible steel frame and which shuts up from a length of twenty centimetres to that of about five centimetres as the wire drill perforates the limb. This transfixion which is done just above the condyles, is very quick and easy, and requires only the use of a local anæsthetic. The wire being in place, a small dressing is applied to its point of entrance and emergence, and a disc-shaped metal shield is clamped on to the wire close to the dressing, so as to prevent the wire from sliding one way or the other when pulled upon. A very strong steel horseshoe-shaped clamp is then made to grasp the two projecting ends of the wire. One limb of the clamp is fixed to the wire by tightening a nut and the other end is then forced towards its fellow by a special screw apparatus which pulls the wire taut. When sufficient tension has been obtained, the second limb of the clamp is fixed by a screw nut to the wire and the ends of the wire are cut off. From the centre of the horseshoe which lies below the knee, a hook, cord and weight are attached and the necessary traction is applied. The appliance is made in two smaller sizes, suitable for use in transfixing the *os calcis* or the olecranon.

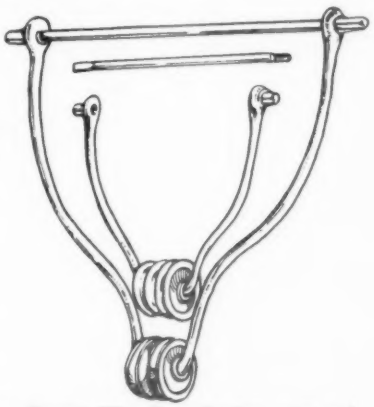

FIGURE III. Transfixation pins and attachment stirrup. Large size for femur. Small for tibia or *os calcis*.

This wire transfixion has one great and outstanding advantage. It is much less liable to cause a pin sinus than any other method of skeletal traction. But, on the other hand, the appliances and technique are complicated and quite unsuited for occasional use by one who has not been accustomed to it. If used for a cancellous bone and especially if this has become soft and decalcified by disuse or disease, the wire will certainly cut its way through the bone and this has to be carefully watched.

Pin Traction with the use of Multiplying Pulleys.

For my own part I think that the original transfixion pin introduced by Codivilla and generally attributed and named after Steinman remains as far the most practical and useful form of skeletal traction. Whilst in principle the pin traction method has been unchanged since its introduction twenty years ago, yet various details in technique have been added or modified and it may be useful to describe these before discussing the indications for the use of this treatment. The pins are made of hard steel in two sizes (fifteen centimetres or six inches long and 4·5 millimetres or three-sixteenths of an inch thick, and 11·25 centimetres or four and a half inches long and three millimetres or one-eighth of an inch thick), and are fitted with a simple handle for their insertion. The larger sized pin is used for the femur or for the malleoli, the smaller, for the *os calcis* or the olecranon (see Figure III). Although the actual insertion of the pin can be done under a local anæsthetic, it is better to give full general or spinal anæsthesia for all healthy patients, because this provides full muscular relaxation, lasting until the patient has been returned to bed and the traction force adjusted. One small but important detail in the care of the transfixion pins is that no pin should be used without having its cutting point freshly sharpened. The use of a blunt pin makes transfixion difficult and tedious and damages the tissues unnecessarily. As regards the best sites for transfixion, in the femur the aim should be to transfix the lower part of the shaft above the condyles, because the bone is dense and will hold the pin more firmly than can be done by the cancellous tissue of the condyles. Transfixion of the crest of the tibia should be just below and behind the tubercle and is the most ideal site of any, because the bone is dense whilst the soft parts traversed are only skin and subcutaneous tissue. In this situation only a small pin is required. The tibial transfixion is suitable for all cases of fracture of the femur, but particularly for those affecting the lower end and the knee joint (see Figures IVA and IVB). The fact that the pull is made from the upper end of the tibia is all to the good, because to this bone are attached both the quadriceps and hamstrings, the contraction of which has to be overcome. The pulling upon the ligaments of the knee joint too is an advantage, because it separates the joint surfaces and tends to prevent adhesions (see Figures VA and VB).

Transfixion at the distal end of the leg can be made through both malleoli, through the tibia alone, just in front of the fibula above the *os calcis* or through the posterior process of that bone. The two former

have nothing to recommend them, and the choice therefore lies between going above the *os calcis* or through it. In simple cases, when the pin is not likely to be kept in place for more than two or three weeks, the transfixion of the bone is simpler and therefore more satisfactory. Placing the pin above the bone is an idea greatly popularized during the war by Finochetti's stirrup. And it is quite good, provided one precaution is taken, namely, the firm fixation of the pin to the foot by means of a plaster of Paris bandage or by some shaped sole-piece. It

FIGURE IVA. FIGURE IVB.

Outline tracings of skiagrams, front and lateral views. T-shaped fracture into the knee joint, showing gross displacement.

is essential that no lateral rocking of the pin in the soft parts should be allowed, because this causes great pain and leads to irritation and inflammation of the skin.

I would reserve the supracalcaneal transfixion for cases in which the *os calcis* is atrophic or decalcified and those in which very prolonged traction is required.

When the pin has been inserted and the point of entrance and emergence covered with dressing, the pin is grasped by a U-shaped stirrup which catches over the head and the point. This stirrup is

provided with two pulley wheels which allow for the necessary multiplication of the weight. The limb is then slung to a Balkan beam or in a suspended Thomas splint or cradle splint. If a Thomas splint is used, it should have the hinged attachment at the knee, so that the leg should always be put up with the knee semiflexed and the foot dorsiflexed. Traction of the transfixed *os calcis* will automatically dorsiflex the foot and in other cases this position can most conveniently be obtained by

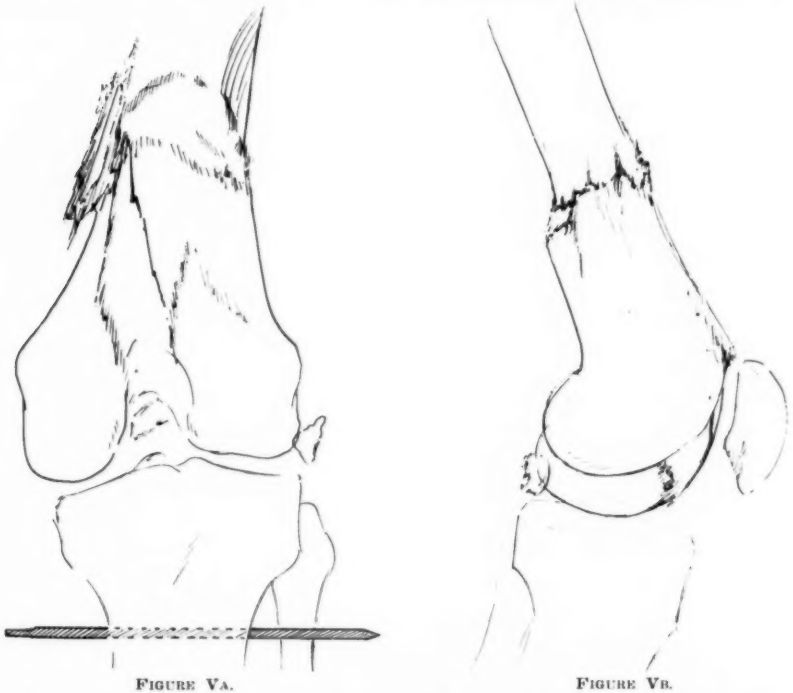

FIGURE VA. FIGURE VB.

Same case six weeks later. After 18 kilograms (40 pounds) traction by transfixation of tibia for four weeks. Excellent functional result.

suspending the toe of the stocking to an appropriate point in an overhead bar. For the application of the traction force a pulley block with two pulley wheels is necessary at the lower end of the bed, and this may be incorporated in the cradle splint. A 4·5 kilogram (ten pound) weight is used, and by employing one, two, three or four of the pulleys a traction force of 4·5, 9·0, 13·5 or 18·0 kilograms (10, 20, 30 or 40 pounds) is obtained. This is much simpler than having to use heavy different weights. I am quite convinced that failure in the application of traction methods is usually due to one of two causes. Either the weight is not

applied directly to the bone and any adhesive appliance attached to the skin does not effects its purpose, or, if skeletal traction is used, the weight employed is insufficient.

It is much better to overdo rather than underdo the weight in the first place. If the length of the limb is checked by measurement and by the X rays, no harm is done; but quite the contrary, if the bones are really distracted so as to be pulled quite clear away from one another.

FIGURE VIA.

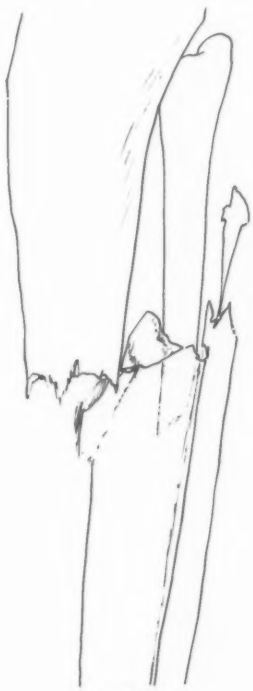

FIGURE VIB.

Tracing of skiagrams of fracture of the tibia and fibula, front and lateral views. Marked lateral displacement.

This has the effect of disentangling the soft parts from the sharp fragments of bone and the latter then slip into place when the weight is lessened.

But, however effectively axial traction is applied, whether by adhesive appliances or skeletal traction, there will always occur some cases in which an appreciable degree of lateral displacement still persists (see Figures VIA and VIB). This may actually spoil the final result and it will certainly delay strong union of the bones. To correct this lateral

displacement one of two methods may be brought to bear on the ends of the bone by suitable rubber bands or by pressure pads attached to the sides of the frame splint by adjustable screws. If they do not succeed, then the seat of fracture should be cut down upon and some direct adjustment and fixation effected. If the fracture is transverse and not comminuted, it should be fixed over a bone peg of suitable diameter. This is an easy procedure if adequate traction has already been applied, so that the fragments are readily separated from one another. If the

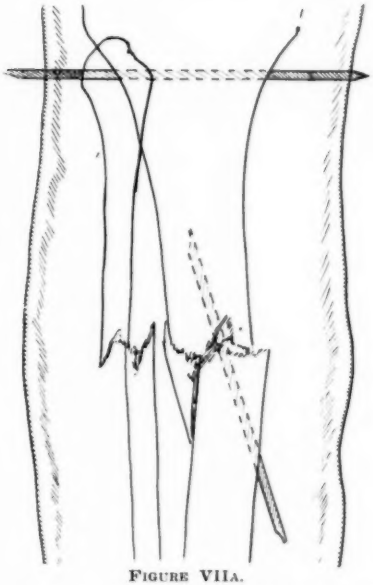

FIGURE VIIA.

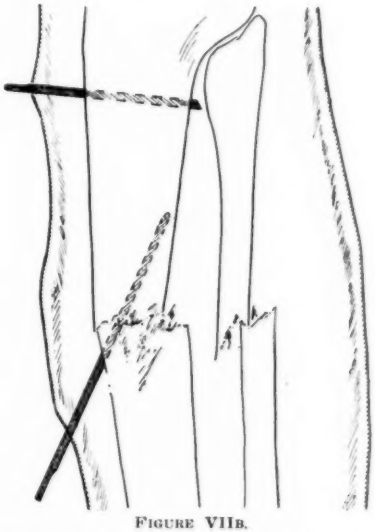

FIGURE VIIB.

Same case after open reduction and pin transfixation with plaster of Paris. (Shaded line indicates the outline of the plaster case.) Excellent functional result.

fragments are oblique with comminution, then when the end-to-end adjustment has been made, this is maintained either by driving a transfixion pin through both fragments or by using two pins or bradawls holding the fragments in position whilst the wound is sutured and finally covering the limb in a plaster case which incorporates the pin or pins (see Figures VIIA and VIIB). These pins can be left in place for one to three weeks, and then are withdrawn without cutting the plaster. This is really a simple modification of the principle of Lambotte's external fixateur in which a number of long pins are screwed into the bone and fixed outside the limb to a strong steel bar.

Another modification of the traction method combined with the use of transfixion pins may be mentioned here, although it is of especial use in the treatment of open fractures. It is most useful in the case of

fractures of the leg bones. A Hawley's table is used, a transfixion pin being put through the *os calcis,* and by this a powerful pull is made on the leg. The seat of fracture is exposed and the fragment adjusted after the removal of intervening soft parts. When persistent displacement still occurs, this is corrected by a second pin put through both fragments. A third pin is used to transfix the crest of the tibia, just below the tubercle, and the whole leg is encased in plaster. This is very much simpler than any plating or pegging operation and, what is much more important, it in no way interferes with the natural process of repair by stripping the bones or devascularizing them. The pin which transfixes the fracture itself is removed in two weeks and others in four weeks.

THE USE OF THE THOMAS SPLINT IN FRACTURE OF THE LONG BONES

By MEURICE SINCLAIR,
London.

THE indifferent treatment of fractures of long bones is a feature of surgery often recognized, but rarely remedied. The careless attitude of the majority of surgeons towards these major injuries is responsible for considerable economic loss, let alone the distress of the patient both during and after treatment. For this reason one welcomes such an article as that published by Mr. C. E. Corlette in March, 1930, in this journal. If fractures are treated as emergencies, if they are submitted to adequate extension having full regard to the anatomy and physiology of the parts involved, there should be a great improvement in the results obtained. Attention to detail is essential, but it does not require mechanical genius to perform the adjustments necessary to produce the good anatomical alignment that is requisite in almost every case if function is to be fully restored. The basis of all treatment in the early stages lies in immobilization, which can be obtained effectively only by extension; this for the greater comfort of the patient is supplemented by suspension.

These principles have always been taught by me, and in no way have I altered them since I commenced to treat fractures during the war fifteen to sixteen years ago. In my book, "The Thomas Splint," I have detailed the methods I use and have amplified them further in my book on fractures published this year. I have no wish to appear unduly critical of Mr. Corlette's article, but there are one or two points which tend to cause confusion in the minds of the reader.

The nomenclature of splints is made difficult by the fact that a splint with a given name is used in a modified method by different surgeons. Mr. Corlette himself has designed a useful splint which follows the apparent general principle of a Hodgen, but is supplied with a hinged joint, spreaders, rings for suspension *et cetera*. My only comment on the description of this splint lies in the realization that a more simple and useful splint could be adapted equally well to his requirements. The splint that I suggest and always use is the one he so emphatically condemns—the Thomas splint—not always used in its original manner, but a splint that can be used throughout treatment of any lower limb fracture.

There are several ways of using the Thomas splint and in fractures of the femur I make use of them all. Firstly, the Thomas splint can be used in its original form, particularly in first-aid emergency treatment. The padded ring takes pressure from the *tuber ischii*, while the fracture is immobilized by fixing the ankle or foot firmly to the end of the splint. The first-aid measures for taking a strong pull from

the ankle or foot are impracticable for proper and more permanent extension. Secondly, the splint can be used in the Hodgen manner: that is, the limb below the fracture is fixed to the foot of the splint, which is in turn pulled on by the extending force, counter extension being made by raising the foot of the bed adequately. The ring of the splint should fit snugly against the *tuber ischii*, but tends to be pulled away from it by the extending force. If necessary, a flexing iron is attached to the side bars of the Thomas splint, at the centre of movement of the knee joint to permit of leg flexion. Tapes or simple knots at the four corners of the splint attach it to the careful balance of suspension weights. If the splinted limb is well balanced, there is no difficulty with nursing and, except for a handle which the patient can grasp with his hands, further lifting apparatus is unnecessary.

In the later stages of treatment I have illustrated the way in which a patient can get out of bed into a chair by this dual method of extension-suspension. The limb itself is slung between flannel strips 10 centimetres (four inches) wide in the case of the leg and 6·75 centimetres (two and a half inches) wide in the case of the arm, and I feel that these are of far more value than the full length sling that Mr. Corlette has devised. In the case of a fracture of the shaft of the femur it is essential to be able to adjust the band if the anterior bow of the femur is to be restored and maintained, and I have yet to see pressure sores resulting from these bands. Fixation of any strips or sling to the side bars of a splint is facilitated by the use of strong paper clips whose adjustment is very easily performed. I discarded safety pins and the like for these clips in the year 1916.

Again, the Thomas splint can be used in comfort as originally designed in the later stages of treatment of a femoral fracture when powerful extension is not required, and also in treatment of fractures below the knee. The pressure of a well-fitting ring is easily tolerated, and suspension again renders nursing simple. A knee flexing iron attached to the side bars renders movement at the knee joint possible under light extension if so desired.

Finally, there is the walking caliper, which surely embodies all the original principles of the Thomas splint. The body weight is carried through the heel *riâ* the side bars to the ring, which presses against the ischial tuberosity. The measurements of the ring and the length of the side bars must be made accurately, for it is my custom to convert Thomas splints into walking calipers for my patients with the assistance of a mechanic, who has simply to cut off the side bars and turn them in to run into a ferrule put into the heel of the boot at the points I mark out for him. The value of this instrument cannot be under-estimated when it is realized that patients can walk about without bearing weight through the leg in comfort for many months and even years.

Admittedly it takes a week or ten days for the skin over the ischial tuberosity to harden at the point of pressure, but ulceration is almost unknown, and complaints other than those of annoyance at having to wear an instrument at all are rare. The tuber-bearing principle ought to be present in most artificial limbs, either above or below the knee

joint, and I stress this point to refute the criticisms that Mr. Corlette has levelled against the Thomas splint as a whole. The ring can and does take pressure efficiently when so required. I must admit that in the modifications the difference in our methods amounts to a quibble, but I maintain that there is no splint devised which can as satisfactorily be used the whole way through treatment of a fractured limb with such success as the Thomas knee splint. I have had no cause to make any alterations or modifications since I first fully understood the principles which underlay the use of this instrument. By its early application and constant use in the Great War, it reduced the mortality from 80% in gunshot wound of the femur in 1916 to 7·3% in 1918.

In the arm the case is somewhat similar. By fitting a swivel ring instead of a fixed one to the Thomas arm splint, it is possible to use it for fractures above the wrist which can be treated in the early stage with a straight elbow. With suspension and extension the so-called "Hodgen" principle again becomes apparent, but the splint remains the swivel arm Thomas splint.

I am glad to find that Mr. Corlette is a strong advocate of skeletal traction, for I have long insisted that any pull from strapping or glue from a limb to total more than 5·4 to 6·7 kilograms (12 to 15 pounds) will flay the limb at the points of greatest pressure.

The Schmerz spring tractor is an excellent instrument of the ice-tong pattern, but Mr. Corlette has several minor complaints to find with it. These, I think, are overcome by the ice-tong caliper designed by Professor Einar Key, of Stockholm, and made by Stille. This tractor is made of stainless steel and has long fine points whose angle is well adapted to the femur. The spring tending to approximate the prongs is powerful, but as soon as the caliper is adjusted it may be held by a simple screw device to prevent the points from forcing their way further into the bone.

When an instrument of the pin or rod type is required to transfix bone, it is advisable to have it as narrow as possible and to leave as little as possible of the outside of the pin to be withdrawn through the bony tunnel when the time arises. The value of the Kirschner "thread" has been recognized for some years in Central Europe, and the instrument is growing in popularity in Britain. It consists of a piano wire driven with a motor or hand drill through the bone in the course of a few moments and then attached to a stirrup in which it is made very tense by a screw. When it has to be withdrawn the stirrup is unscrewed, a projecting end is cut off and the wire pulled through easily and painlessly. My objection to it is that the projecting end cannot be sterilized before being withdrawn.

In my own practice I have a number of instruments for producing skeletal traction and have illustrated them in my books, where I give my reason for preferring them to other appliances.

Open operation on fractures is, as is commonly understood, applicable to certain fractures of necessity, but in other cases it must not be resorted to until the effects of extension have been tried. If,

owing to interposition of muscle or the like between fragments, I have to operate, I always put in a small internal splint (wire or plate, or even linen thread for the patella, all of which can be sterilized by boiling) after reduction in addition to maintaining extension. The value of this internal splint is considerable, even if it has to be removed at a later date, since it supports the fracture against strains such as gravity, muscular contractions, and prevents slipping of fragments which may occur from loss of substance on one face of bone. Also an internal splint will, for example, resist injudicious increase or relaxation of extension when a patient is being transferred to and from the operating theatre. Still, open operation on fractures must be undertaken only under absolute aseptic technique, as damage done by sepsis is irrepairable and is catastrophic. Other conditions are that the replacement of fragments should be done through a small incision, without unduly disturbing the anatomy of the parts, the position obtained must be anatomically and radiographically perfect and nothing short of it—otherwise you have failed in what you have set out to do. Finally, this reposition must be maintained until the union is solid, not only for a few hours or weeks, but sometimes it must be preserved for many months. Under these conditions open operations are fully justifiable.

In compound fractures I always deplore excision of apparently infected bone, which causes loss of substance which cannot be fully restored. I much prefer to clean up the wound, excising grossly infected or mutilated soft tissues, and then draining the area by gravity—a procedure of simplicity in the case of a fracture treated in a Thomas splint. The risk of leaving infected tissue in the wound in cases which are submitted to excision and primary suture is too great, since gas gangrene and tetanus have often caused loss of limb and even life following this treatment. During the war the efficacy of Carrel-Dakin treatment was frequently tested and commented on, but in my hospital we had a ward set aside for the Carrel-Dakin treatment until it was noted that sequestration was out of all proportion, both in number and size of sequestra, to clinically similar cases treated with dry dressing only. Cases treated simply with dry or moist saline dressings did better than those treated carefully and correctly with the Carrel-Dakin or any other form of irrigation. This was so definite, and prolonged trial established the point to such a degree, that I have used only dry or saline dressings since that time. But I always drain the wound by gravity, so as never to allow any pus to stagnate in the tissues. The temperature and pulse of a patient with a compound fracture, if the fracture is properly immobilized and drained well, in the very large majority of cases return to normal by lysis on the tenth day.

The purpose of this short article is in no way intended as a criticism of Mr. Corlette's excellent paper, since I heartily endorse most of that which he has written, and only wish that such doctrines as he has taught could be followed more widely. My comments are meant only in the nature of amplifications and in certain instances to prevent misapprehension which tends to arise from our complicated nomenclature and varied teaching.

EXPERIMENTAL TRANSPLANTATION OF URETERS INTO THE INTESTINE.

By P. N. WALKER-TAYLOR.

[From the Department of Urology, Royal Prince Alfred Hospital, Sydney.]

SINCE the middle of last century the establishment of a safe method of transplanting the ureters into the bowel has been the aim of many surgeons.

INDICATIONS.

Originally the operation was conceived for the relief of patients suffering from exstrophy of the bladder, a condition stated to occur once in 30,000 to 50,000 births.[1] Statistics show that of these patients 50% die largely as the result of neglect[2] before they reach their tenth year and two-thirds before they reach their twentieth.

The following further indications have been mentioned:[3] (i) Incurable carcinoma of the bladder, urethra and prostate for palliative purposes; (ii) advanced carcinoma of the bladder, urethra or prostate in which it is impossible surgically to remove the growth, but in which it is possible with the use of large doses of radium to destroy it; (iii) early curable carcinoma as an essential part of the operation of total cystectomy; (iv) carcinoma involving the urethra; (v) incurable vesico-vaginal fistula; (vi) contracted bladder due to scars of ulceration or due to other causes; (vii) tuberculous ulceration of the bladder in which one kidney remains good, the other to be removed; (viii) traumatic injuries which make the use of the bladder impracticable; (ix) some cases of carcinoma of the *cervix uteri*, in which adequate radium treatment demands total vesical exclusion.[4]

HISTORY OF THE OPERATION.

No attempt is made in this article to give a complete survey of the history of the operation. I have merely selected here and there the names of workers whose investigations represent progressive steps in the improvement of the procedure. Kirwin[5] in 1930 presented a fairly comprehensive monograph on the history of the subject and on this I have largely drawn.

The first attempt at establishing a communication between the ureter and the bowel was made by Sir John Simon on July 5, 1851.

Instruments of a very ingenious construction were contrived for Simon, by means of which he was able to pass threads from the ureters, as they lay exposed on the ectopic bladder, to the adjacent rectum. Traction upon these threads induced pressure necrosis and eventually uretero-rectal fistulæ. Some twenty days later most of the urine was flowing into the rectum and in the course of three

months the operator sought to close up the natural ureteral openings and protect the exposed bladder. The patient died of exhaustion in about a year. Autopsy showed great disorganization of the urinary apparatus due to "suppurative inflammation."

Lloyd in the same year attempted a similar procedure; but in his case the needle passing from the ureteral orifices to the rectum traversed abnormally placed peritoneum and peritonitis ensued which proved fatal on the seventh day. Actual transplantation of the ureters was first performed by Thomas Smith in 1878. Through a left lumbar incision the ureter was cut as low as possible and passed into the descending colon through a small incision. A single suture of catgut through the outer layers of ureter and bowel was employed to anchor the ureter in position. After six days urine and fæces appeared in the wound. The fistula, however, closed in a month and the patient appeared in good health. In one year's time a similar operation was performed on the right side. Death from uræmia followed within fifty hours. The right kidney was the seat of acute pyelonephritis, whereas on the left all the secreting tissue had been destroyed long before by infection. Smith concluded that "any communication between ureter and bowel was, in itself, a fatal lesion."

Kuster in 1891 transplanted both ureters into the rectum after removal of a carcinomatous bladder. Death followed in five days from peritonitis and renal infection.

Chaput in 1892 performed a unilateral transplantation for ureterovaginal fistula. The patient lived, but no further details of this case could be obtained and, as the operation was unilateral, the history is inconclusive. In a second case—a double implantation—the patient died of uræmia. In the next three years three attempts were made with fatal issue.

Karl Maydl in 1894, conceiving that by retaining the ureteral orifices intact regurgitation would be prevented and thus ascending infection obviated, resected the entire trigone and implanted this into the neighbouring sigmoid. This operation was successful and the patient was in good health seven years later. Maydl operated upon nine other patients by this method, which was also adopted by other surgeons.

Buchanan[6] in 1909 collected 80 cases operated upon by the Maydl method (trigono-sigmoid implantation), of whom 23 died as an immediate result of the operation. He adds that if modifications of this method were added, the mortalities would be much higher than this 28·7%.

Moynihan modified this operation to some extent in 1905, but employed the same principle.

In 1894 also B. Bergenhem[7] in Sweden implanted the ureters separately into the rectum of a man by the extraperitoneal route. He removed all the ectopic bladder wall, except an oval piece surrounding each ureteral orifice. He then freed the ureters by blunt dissection, with every care to avoid opening the peritoneal cavity. This was accidentally opened in two places and immediately sutured. The rectum

was exposed and opened by two very small antero-lateral incisions, through which the corresponding rosettes of bladder, carrying the freed ureteral seeds, were drawn.

The second operation of this nature was performed by Jaja, of Florence. This was actually performed before Bergenhem's operation, though published later. The third was performed by Pozza, of Italy, 1897; the fourth by Martin, of Chicago, 1898; the fifth by Capello, of

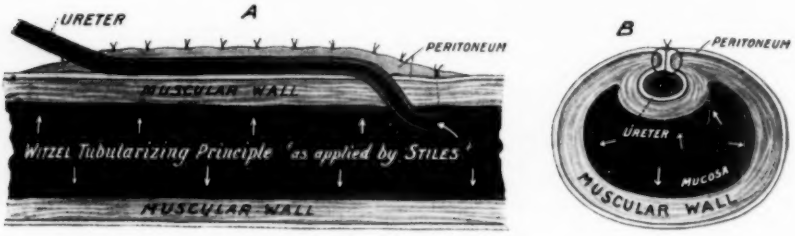

FIGURE IA. Showing the Witzel tubularizing principle as applied by Stiles. (After Coffey, *Surgery, Gynecology and Obstetrics*, May, 1921.)

Rome, 1898; the sixth by A. A. Lendon, of Adelaide, Australia, on May 12, 1899; the seventh by George A. Peters, of Toronto, Canada, on July 5, 1899. This is the operation that is known throughout Great Britain and America as the Peters operation. Buchanan in 1909 collected histories of 26 patients on whom this operation had been performed with three deaths—a mortality of 11·5%. An astomotic button

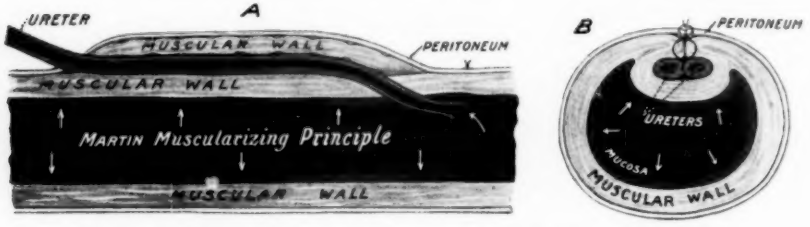

FIGURE IB. Showing the Martin muscularizing principle. (After Coffey, *Surgery, Gynecology and Obstetrics*, May, 1921.)

was used by Boari, an Italian, in 1894, and a hollow nickel tube by Chalot in 1896. The results reported, however, are inconclusive where not fatal.

Fowler, in America, in 1896 presented an entirely new plan. Through a mid-line abdominal incision the ureters were severed close to the bladder. The rectum was incised longitudinally through serous and muscular coats, which were then retracted exposing a diamond-shaped area of mucous membrane. A flap of this mucous membrane was cut with

its base upwards. This was folded upon itself and sutured to form a valve having mucous membrane on both sides. The ends of the ureters, cut obliquely, were sutured to this flap, which was pushed into the

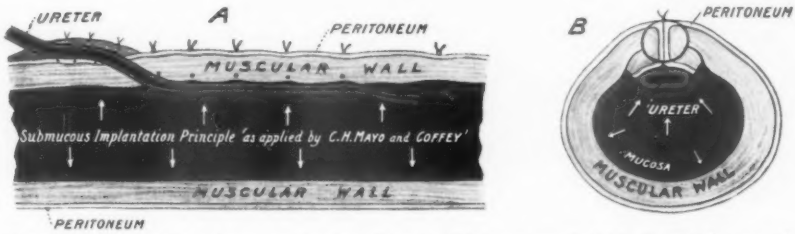

FIGURE IC. Showing submucous implantation principle as applied by C. H. Mayo and Coffey. (After Coffey, *Surgery, Gynecology and Obstetrics*, May, 1921.)

lumen of the rectum, the opening of which was then closed. This is the flap-valve of Fowler which is intended to close the ureteral openings with the descent of the bowel contents. Fowler reported success in one case, but other operators were not so successful, several unilateral operations being inconclusive.

FIGURE ID. Showing Fowler mucous membrane flap. (After Coffey, *Surgery, Gynecology and Obstetrics*, May, 1921.)

Two other names must be mentioned: Martin and Stiles. So far all the methods described have been of the "direct implantation" variety. That is to say, that the

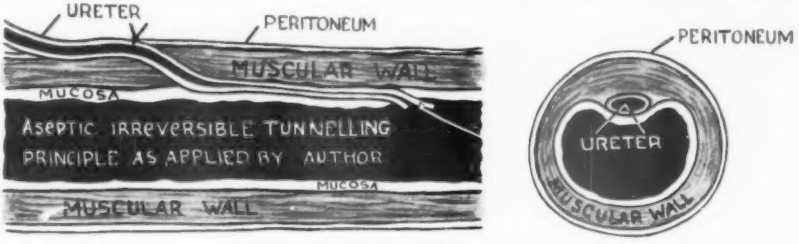

FIGURE IE. Showing author's operation.

ureter meets the bowel and enters the lumen directly or perpendicularly. The work of these two men embodies a new principle: that of indirect or oblique implantation. As Martin[8] says in enunciating the principles

underlying his experiments: (i) The ureters should empty into the bowel in a longitudinal direction from above downwards. (ii) The ureters are buried in the walls of the rectum for a distance of an inch or more longitudinally before entering the lumen, so that in the act of defæcation the fæcal mass will close the calibre of the ureters by its pressure on the mucous membrane.

Briefly, the method of Stiles[1] is an intra-peritoneal anastomosis of the ureters into the recto-sigmoid. About one and a half inches of the ureter are buried in the bowel wall, much after the style of a Witzel gastrostomy.

The obliquely cut end of the ureter is passed through a small transverse incision in the longitudinal muscular band of the bowel at the spot selected into the lumen of the bowel and anchored within the bowel lumen 1·25 centimetres (half an inch) lower down by a catgut suture which follows a needle out through the bowel wall at this point. The process of burying the ureter is now carried out by a series of interrupted sutures which take a good hold of the bowel on either side, but at such a distance that, when drawn over the ureter, the latter has ample room to lie in the groove so made without fear of causing its constriction. A second and final row of sutures buries the fixation stitch and the first row of inverting sutures. Grey Turner[1] operated upon sixteen patients by this method with four deaths.

Martin's method is similar, except that he first incised the bowel peritoneum longitudinally and retracted it from the muscle. The ureter is now implanted through a distal stab wound and the ureter is buried by invagination of the muscular layer. Finally the peritoneum is oversewn. Martin operated upon four patients. In three death followed almost immediately; in the fourth only one side was done. Martin did a great deal of experimental work on animals, but finally relinquished the attempt to find a clinically satisfactory operation, and stated that he was convinced that work along these lines would never be crowned with success.

When I commenced an experimental study of the subject in October, 1929, a perusal of the literature seemed to indicate that the method devised and practised by Robert C. Coffey, of Portland, Oregon, United States of America, was outstanding in offering a greater likelihood of universal success both from a theoretical point of view and also from the results produced by Coffey himself and other operators.

The description of Coffey's original technique[9] was first read before the Section of Surgery of the American Medical Association in June, 1910. He presented six dogs operated on by the method of direct implantation. Of these, all the dogs had died except one. In the one which lived the ureter was somewhat dilated and very much thickened and the kidney had been destroyed, leaving a fibrous knot. The opening into the bowel was large and the ureter was a diverticulum on the large intestine. He presented nine dogs operated upon by his new "submucous" method. Three died of peritonitis, one died thirteen days later from no discoverable cause; five recovered and lived for periods of seventy-two to one hundred and sixty-nine days after operation, when

they were killed with chloroform. He stated that in none of these cases did ascending infection take place. The record does not state whether a double implantation was performed or only one side. Coffey stated: "This was the first series of consecutive successful implantations of the ureter into the large intestine of dogs that had been reported." He described his original technique as follows:

> The duct is cut . . . and one wall split down with a pair of scissors. A linen suture is passed through the split end and tied. The loose ends are threaded into two needles. . . . The part of the intestine desired is picked up and an incision made down through the peritoneal and muscular coats, including submucous tissue. The muscular layer is loosened from the mucosa with the point of the knife until the mucous membrane pouts out through the incision. This incision should be about one inch long or more. Then five or six sutures are passed which pick up the peritoneal and muscular coats on each side of the incision. The intermediate intestinal sutures are lifted upon the flat handle of an instrument as they cross the incision. Now the intestine is brought down close to the end of the split duct and the two needles carrying the threads (traction sutures) on the end of the duct, are passed beneath the four or five intestinal sutures and through a stab wound in the mucous membrane, at the lower end of the exposed mucous membrane, into the intestinal lumen and out through the intestinal wall three-quarters of an inch farther along the intestine and one-eighth to one-quarter of an inch apart. By making the tension on these threads and at the same time pushing the intestine towards the duct, the duct is drawn beneath the intestinal sutures, through the stab wound into the intestinal lumen. The two ends of the threads on the duct are tied on the outside, thus anchoring the end of the duct on the inside of the intestine at this point. The intestinal sutures are then tied. . . . It is necessary to tack the ureter to the peritoneum of the intestine near its point of entrance by two or three fine linen or silk sutures.

Charles Mayo[10] modified this technique thus: he used catgut instead of linen or silk. He inserted the sutures approximating the cut muscle and peritoneum after the ureter was anchored, and with each alternate stitch caught a bite of the outer wall of the ureter. He used another row of continuous stitches over this first set to give additional protection. His traction suture on the end of the ureter consisted of a single strand; the other end of the suture, being cut about three inches long, he coaxed up the lumen of the ureter. This is the "catgut urine guide" of Charles Mayo and its intention is to maintain the patency of the ureter and to facilitate drainage of urine during the first few days after operation.

It will be noted that a duct entrance has been established similar to other natural duct entrances in the body; for example, the normal ureter or the bile duct. By this arrangement the ureter, being separated for some distance from the bowel lumen by the mucosa only, is subjected to the "intraintestinal pressure" laterally applied. This pressure keeps the ureter collapsed in between peristaltic contractions.

In "Osler's Memorial Volumes" (1919)[10] Dr. Mayo makes the following report of his work on exstrophy of the bladder:

> 52 patients seen.
> 6 had plastic operations: 1 died six months later.
> 3 operated on by Maydl-Moynihan technique: 2 died of uræmia.
> 26 operated on by transplantation method: 4 died as result of operation.
> 17 not operated on yet.

G. Grey Turner quotes the following Mayo Clinic statistics from 1901 till April 1, 1926:

Number of patients operated upon	71
Plastic operations only	8
Transplantation of both ureters	46
Transplantation of one ureter	10
Exploration only	2
Moynihan's operation	2
Coffey's operation	3

That is to say, 61 patients had a transplantation operation performed, 10 of these on one side only. There were eleven deaths from the operation: five from peritonitis, four from septic kidney, two from renal insufficiency. There were seven late deaths from two months to two years after the operation; only one was due to renal complications.

The fact that during the past twenty-one years since 1910 no new principle has been enunciated, while Coffey's technique is now almost universally employed, seems to indicate that surgical opinion has judged the principle of "submucous transplantation" to be correct, but the proved and safe final modification is yet awaited.

Modifications of the original technique, generally in respect of minor details, have been published from time to time; but the two most important and striking contributions have come from Coffey himself.

Thus Coffey has developed three plans. He writes:

> In plan A [the one described above] the immediate operative results on dogs were bad. According to the best information I can get, this plan of operation has been used in approximately 200 clinical cases by many operators. Official or unofficial reports indicate that the operative mortality has been 25% or more. Nearly all patients who recover, however, have a stormy convalescence because of local infection entering the wound by the opening in the mucosa through which the ureter has been drawn.

Plan B is the tube technique described in *Surgery, Gynecology and Obstetrics* of December, 1927:[11]

> The new features introduced are the thorough cleansing of the bowel and the use of ureteric catheters. Preoperative treatment consists of light diet, castor oil administration on two successive days prior to operation, flushing of the bowel with high enemata on the day before and the day of operation. The abdomen is opened and a rubber covered stomach clamp placed on the sigmoid 12 to 14 inches above the anus. A sigmoidoscope is inserted into the rectum. A tube two feet long in which has been fastened a bevelled edge needle, is coupled to the tube of an irrigator which is arranged at a considerable height. The needle is thrust through the wall of the sigmoid just below the clamp and the water is turned on. As soon as the water flows clear, 1% mercurochrome is put in the irrigator. After the irrigation is finished, the obturator is again inserted and the sigmoidoscope is pushed upward to within an inch of the obstructing clamp. Sterilized gauze is now used to pack the lower bowel with the aid of laryngeal forceps. As the bowel is filled with gauze, the sigmoidoscope is gradually withdrawn.
>
> The two ureters are cut low down and mobilized for a couple of inches. A ureteral catheter is now inserted into each ureter down to a rubber cuff which has been previously fastened on the catheter six or seven inches from its tip—this refers to the human case. Two strong double linen ligatures are tied around

the slit portion of the ureter and around the anchor cuff. A third linen suture is placed around the ureter above the cuff.

Attention is next turned to the intestinal part of the operation. The lowest point in the sigmoid at which the operation can be conveniently done is considered to be the site of election. Two incisions are made diagonally about an inch and a half in length, extending from the mesentery's edge downwards and toward the mid-line of the intestine. The left incision is made lower than the right. The knife carefully makes each incision through the peritoneal and muscular coat which is pushed off the mucosa. A stab wound through the mucosa at the lower angle of the incision exposes the gauze. To this gauze the free end of the corresponding catheter is sewn on each side. Withdrawal of the gauze from the anus draws the catheters and finally the ureter ends through the stab openings in the mucosa and into place within the lumen of the bowel. The cut peritoneum and musculature are reunited over the ureter by sutures, several of which take a bite of the ureter. Another line of sutures may be used to cover the first line.

Using this technique, together with a system of retro-peritoneal drains, Coffey operated upon nine consecutive patients with no fatalities. There was one ureteral leak and one ascending kidney infection. Renal function was seemingly undiminished. Coffey writes:

In plan B the technique is very exacting, but it is far less dangerous than plan A. The sepsis around the ureters in the intestinal wall is much less, and the post-operative reaction and immediate mortality are much less than with plan A. Plan B includes such an extensive and exacting technique that it is practically impossible to use it on dogs. The mortality during my own experiments on dogs was high by this technique.

Plan C Coffey first described in March, 1930, in a preliminary report. He again described it thus in *The Journal of the American Medical Association*, May, 1930:[12]

The ureter is mobilized in the usual manner and cut between forceps. . . . A curved needle, carrying 00 chromic catgut doubled transfixes the ureter just above the forceps. The loose end of the suture is left long and tied both ways around the ureter. An incision is then made in the intestinal wall about one and a half inches long, beginning near the mesenteric border and extending inwards and downwards towards the antimesenteric border. The incision as usual is down to the mucosa and the muscle edges are separated. The curved needle carrying the catgut which is attached to the ureter passes between the cut muscular coat and the mucosa from within outward, through the peritoneum and back through the peritoneum between the muscle and the mucosa on the other side. By using the loose end of the suture, one may draw the ligated end of the ureter into the angle of the wound between the mucosa and the muscle. This firmly anchors the ureter. About a quarter of an inch proximal to the ligature a fine straight needle carrying a strong fine linen or silk thread doubled transfixes the ureter in such a way as to include at least half its circumference. The needle then picks up the loose mucosa and includes at least a quarter of an inch of the full thickness of the mucous membrane. This suture is tied tightly so as completely to strangulate the tissues within its bite. The wound in the intestine is closed over the ureter with a continuous suture. The peritoneal edge forming the outer margin of the incision from which the ureter has been lifted is drawn across and sutured so as to cover the ureter entirely.

As the bowel is not opened, the operation may be entirely aseptic, as is proved by the fact that in the seven single ureters transplanted at seven operations, there has been no evidence of adhesions or peritoneal infection in the neighbourhood of the intestinal wound. The urine has

appeared in the rectum at periods varying from twenty-four to forty-eight hours. Coffey comments:

> If the late results of this plan are as good proportionately as the early results, it may prove to be important. It is, of course, possible that the fistula may later close. Therefore much more experimental work must be done before this plan can be recommended as a clinical procedure.[13]

In February, 1931, I. R. Sisk, J. B. Wear and H. A. O'Brien,[14] of Wisconsin, United States of America, after considerable work on this subject, published very interesting results. They summarize:

> In our hands the transplantation of the ureters to the sigmoid in dogs is highly unsatisfactory by the Mayo technique. In every case the kidney became infected and closure of the uretero-sigmoidal opening by the mucosa of the bowel occurred in five out of seven unilateral transplants. In the two bilateral transplants the ureteral openings were markedly stenosed.
>
> The completed Coffey technique [meaning Coffey's tube technique] gave slightly better results. Of the eight dogs, four showed bilateral infection, three showed infection in only one side and one showed no evidence of infection, but was hydronephrotic on one side. Complete closure of the ureteral opening occurred in only one ureter. . . . Some degree of hydronephrosis was found in most of the kidneys. . . . All of these animals were dead as a result of the operation by the thirty-first day. A completely normal kidney was not seen in the entire group. A clinical case showed infection to begin while the catheters were still in the lumina of the ureters.

THE AUTHOR'S WORK.

In my own work, commenced in October, 1929, which comprises 135 operations on 77 dogs with the implantation of 106 ureters, I have adhered to no single technique, but, as the title suggests and as the protocols show, have been experimenting and modifying from first to last.

I commenced by following Coffey's original technique. A high mortality resulted in this series, which was in most instances due to peritonitis arising from the site of implantation. It seemed that infected material found its way from the bowel, between the stitches in the bowel wall, into the peritoneal cavity.

It was obvious that if incision and resuture of the intestinal muscle and peritoneum could be avoided, whilst still retaining the principle of submucous transplantation, then a definite improvement would be effected. It was conceived that burrowing a longitudinal tunnel in the submucous layer of the bowel would comply with these two conditions. This idea was employed for the first time on January 29, 1930, and has been embodied in all subsequent work. The tunnel principle, thought to be impracticable by Coffey,[11] is the feature of our work which essentially differentiates it from the work of all other investigators on this subject.

This principle was first put into effect with the "open tunnel technique" (plan A). This plan was improved upon in the "closed tunnel technique" (plan B). A further modification, introducing the factor of positive asepsis, forms plan C.

Plan A: The Open Tunnel Technique.

Without the Use of Ureteral Catheters.

The ureter is severed and the end split in the usual way. A fine catgut suture on a straight needle is affixed to the cut end. The section of bowel which is to receive the ureter, usually the recto-sigmoid, is picked up and held with the fingers of the left hand just proximal to the site of the proposed tunnel. With the point of a scalpel a small transverse cut about three millimetres (one-eighth of an inch) long is made through the peritoneum and into the muscular layer of the bowel just distal to the fingers of the left hand. Into this incision the blunt dissecting point of the tunnelling instrument is introduced and pushed down longitudinally in the submucous layer between the circular muscle and the mucosa for a distance of 1·8 to 2·5 centimetres (three-quarters to one inch). For a tunnelling instrument we have used a blunt dissector of the type called after Sir Alexander MacCormick, the Australian surgeon (see Figure II). The formation of the tunnel is not perhaps as easy as the above description might suggest; but a little practice in the correct method makes the procedure sufficiently simple and certain. The tip of the instrument is inserted, as stated above, into the small incision and, the correct plane having been found, the instrument is directed

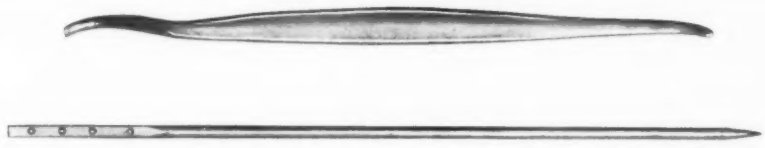

FIGURE II. This is a MacCormick's blunt dissector which was employed in this work as the tunnelling instrument. The lower instrument is a piercing instrument. These drawings are half the actual size.

distally. The tunnel is made by successive applications of moderate pressure, the bowel being securely held proximal to the incision with the fingers of the other hand. This is usually sufficient, but sometimes, in cases of undue difficulty, by a stroking motion of the tip of the instrument towards the lumen of the bowel, the mucosa is by degrees pushed away from the muscular wall for the required distance. Thus we have a narrow blind tunnel about 2·5 centimetres (one inch) long, the diameter of which is probably three to four millimetres (one-eighth to one-sixth of an inch). The bowel lumen has not yet been entered. At the distal end of the tunnel a small longitudinal incision about three millimetres (one-eighth of an inch) long is made through the peritoneum and muscle of the bowel into the tunnel itself. We now have a tunnel open at both ends. Through this tunnel from above downwards is passed the straight needle, carrying the catgut attached to the ureter. The needle is more easily passed backwards so that the point does not catch in the sides of the tunnel. The catgut is pulled through a certain distance, the straight needle is removed and a curved one threaded in its place. The mucosa seen through the distal tunnel opening is nipped with a pair of mosquito forceps, and the peak pulled through into the opening. The mucosa is now opened with a slim knife or scissors. The curved needle is passed through this opening and out through the entire thickness of the bowel wall about eight millimetres (one-third of an inch) lower down. Traction is made on the suture until the tip of the ureter passes into the proximal tunnel opening, down the tunnel, through the perforation in the mucosa and finally to its attachment within the lumen. The ureter is anchored here by the catgut suture. The distal bowel incision and the anchoring suture are wiped with swabs soaked in ether. This incision is closed with one or two fine catgut sutures. The ureter is again anchored by a single catgut stitch passing through the outer layers of the ureter and the bowel muscle at the proximal end of the tunnel. The whole operation area in the bowel is then buried by a longitudinal line of sutures which picks up peritoneum and muscle on either side.

With the Use of Ureteral Catheters.

It is unnecessary to describe all the steps of this modification. Into the cut ureter is tied a catheter which is passed along the tunnel, through the mucosal opening and out the anus through the agency of a rectal cylinder which has been thrust up the rectum by an assistant to the level of the mucosal opening. The ureter is anchored and the remaining steps of the operation are not varied.

By this open tunnel method we operated upon a fairly large series of animals, several of which are still living in perfect health, with both ureters transplanted, sixteen months after operation, or have been used for the purpose of other experiments. With this method, although the risk of leakage of bowel contents between intestinal stitches is much lessened, it is not eliminated. Plan B was then formulated.

Plan B: The Closed Tunnel Technique.

Without the Employment of Ureteral Catheters.

The ureter is prepared in the usual way and a long fine straight needle threaded on the catgut. The tunnel is made, but is not opened at the lower end. The mucosa is perforated at the distal end of the tunnel by a perforating instrument shaped like a pencil (see Figure II), which is distinctly felt to pass through the mucous membrane into the lumen. The instrument is withdrawn and a blunt-pointed director is passed down the tunnel through the mucosal perforation and into the bowel lumen to impinge against the anterior aspect of the bowel wall the determined distance (for example, eight millimetres or one-third of an inch) beyond the mucosal perforation. The straight needle is now passed along the groove in the director to its tip and out through the bowel wall at the point of impact. The director is withdrawn, the incision in the bowel wall is wiped with an ether-soaked swab and the catgut is drawn through the bowel wall until the tip of the ureter reaches its destination within. The end of the ureter is anchored here. A further stitch anchors the ureter at the proximal end of the tunnel if desired, though this was not done in most of our cases.

This completes the operation, unless it is thought necessary to anchor the bowel to the parietes to avoid kinking of the ureter. This, of course, is a universal consideration and does not apply only to this technique. No infolding sutures are used, though the terminal anchoring stitch may be buried.

With the Use of Ureteral Catheters.

The procedure with the use of ureteral catheters is obvious and a description is unnecessary.

Plan B technique makes two advances: (i) It diminishes the extra risk of obstruction to the ureter which the infolding layer is likely to impose in a bowel of small calibre. (ii) It eliminates the risk of post-operative leakage of bowel contents. From the point of view of asepsis, however, it is still far from perfect. There are two sources by which infection is brought direct from the bowel lumen into the peritoneal cavity during the operation itself. Firstly, the terminal anchoring suture on the needle passes from within the bowel directly outwards, and, although this anchoring stitch may be buried subsequently, it has already inevitably infected the bowel wall and the fingers. Other parts also may be contaminated directly, and other parts will almost certainly be contaminated indirectly through the fingers.

Secondly, two instruments, the perforating instrument and the director, are passed into the intestinal lumen and back again through the tunnel into the peritoneal operative field. Although the withdrawal of these instruments may be accomplished with discretion and the bowel incision afterwards carefully wiped with ether-soaked swabs, infection nevertheless must still lie at the entrance of the tunnel, as it inevitably lies in the tunnel itself. The passage of the ureter down the tunnel in the next step mops it out to some extent, but this to-and-fro motion of the instuments certainly vitiates asepsis. If, during the operation, it could be arranged that after the mucosa is opened the terminal anchoring

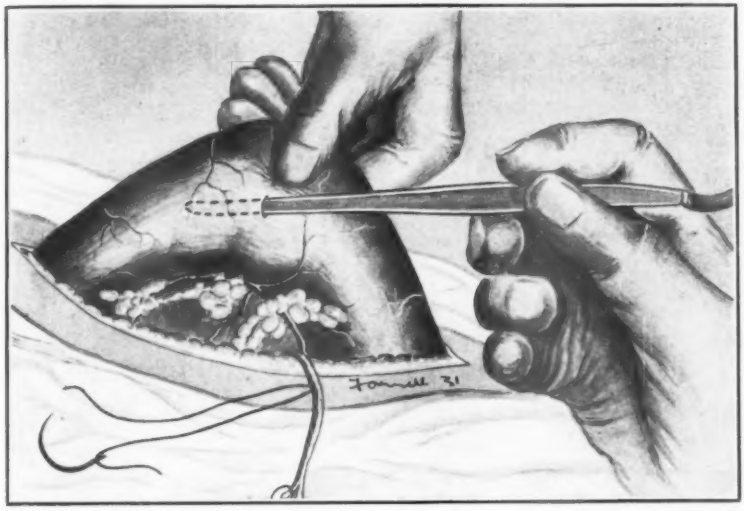

FIGURE III. This indicates the method of fashioning the tunnel with the blunt dissector in the operation employing the aseptic-irreversible-tunnel method.

suture and instruments all pass in one direction (from the peritoneum, along the tunnel, to the bowel lumen, without any returning), then this objection would be removed. This has been accomplished in plan C, which I have called the "aseptic irreversible tunnel" technique. In this operation all instruments pass from an aseptic to a septic field. Infection lies at least eighteen millimetres (three-quarters of an inch) in a practical sense, depending on the length of the tunnel, from the field of operation and, furthermore, once the ureter is in place, has no inducement for creeping any closer.

Plan C: The Aseptic Irreversible Tunnel Technique.

This plan is presented in two forms. Though the same principle obtains in both, the procedures are slightly different.

With the Use of the Piercing Instrument.

Without the Employment of Ureteral Catheters.—The piercing instrument is a straight metal instrument, somewhat like a darning needle, 22·5 centimetres (nine inches) long. One end is pointed like a pencil, the other is flattened for about four centimetres and bears a series of three or four perforations. Several of these must be at hand to match the calibre of the ureter found at operation (see Figure II).

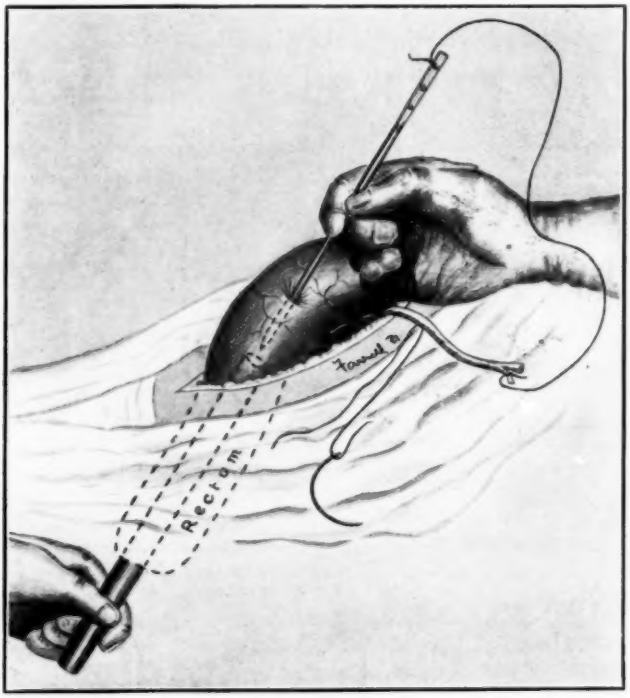

FIGURE IV. This shows the stage reached just before the mucosa is perforated. The piercing instrument is fitted into the end of the rectal cylinder with the mucosa intervening.

The ureter is severed and the end split for six millimetres (a quarter of an inch). A silk suture 45 centimetres (eighteen inches) long is attached firmly to the split end of the ureter, whilst the other end of the ligature is laced through the terminal perforation on a piercing instrument of slightly larger calibre than the ureter and tied. A fine curved needle, carrying fine catgut, is passed through the outer layers of the ureter, or through the periureteral tissues if any adhere, about 3·1 centimetres (one and a quarter inches) from the end of the ureter. This becomes the lateral anchoring suture. Attention is now turned to the bowel, where it is proposed to implant the ureter, usually the recto-sigmoid. A spot is selected on the bowel which lies comfortably and naturally against

the anchoring suture. This spot is taken as the proximal end of the tunnel. A tunnel of 18 to 25 millimetres (three-quarters to one inch) is fashioned (see Figure III). The ureter is measured alongside the tunnel and the tunnel made of such a length that only a little more than six millimetres (a quarter of an inch) of the ureter will project within the bowel lumen in the final position.

A straight metal cylinder about 30 centimetres (a foot) long and 1·25 centimetres (half an inch) in diameter, with a blunt obturator to match, is now thrust through the anus and along the rectum by an assistant until, guided within the abdomen by the operator, it comes to lie at the level of the distal end of the tunnel. The obturator is withdrawn by the assistant and the operator, with the fingers of the left hand, holds the cylinder through the thickness of the intestinal wall at the distal end of the tunnel. The piercing instrument is

FIGURE V. This shows the operation complete. When the bowel is dropped back, the ureter takes a direct course.

introduced down the tunnel until its point makes a tent of the mucosa at the distal fundus. This apex is manipulated into the top of the rectal cylinder (see Figure IV). Whilst the rectal cylinder is firmly held by the assistant the piercing instrument is thrust through the mucosa and down the cylinder. When the posterior end of the piercing instrument has nearly reached the tunnel, the cylinder is withdrawn and the tip of the piercing instrument will be seen protruding through the anus. This is grasped by the assistant and traction is made upon it. Thus the piercing instrument disappears down the tunnel and out the anus. This is followed by the silk suture until finally the ureter reaches the tunnel entrance. Before the procedure is carried further, it is made certain that the ureter is entering the tunnel without any twist; traction is then allowed to continue until the lateral anchoring suture has reached the proximal end of the tunnel (see Figure V). Traction is now stopped and the silk is strapped or

stitched to the skin outside the anus. The ureter is, in addition, anchored by the lateral anchoring suture at the proximal end of the tunnel. Closure of the abdomen completes the operation.

In these experiments we did not cover over the transplantation area with the lateral cut edge of the peritoneum lying superjacent to the ureter bed. The silk cuts through the ureter end and is defæcated within the week.

With the Use of Ureteral Catheters.—This technique is admirably suited to the employment of ureteral catheters. The piercing instrument as described above has several perforations through the flattened end. The end of the catheter, being laced through these, is firmly held as it is drawn down the tunnel and rectal cylinder till the ureter lies in place.

With the Use of the Mucosal Punch.

Without the Employment of Ureteral Catheters.—For the operation with use of the mucosal punch and without the use of ureteral catheters, several special but simple and easily made instruments are required.

The mucosal punch consists of a thin metal cylinder about 12·5 centimetres (five inches) long, sharpened at one end. The shaft of an ordinary cork cutter represents the type of thing required and was, in fact, used in our experiments. To the unsharpened end of the punch is attached (welded) a straight flexible wire shaft about 30 centimetres (a foot) long, looped at the end. Several of these instruments of varying calibre should be at hand to match the size of the ureter found at operation. A blunt metal obturator to fit the punch is required, also the rectal cylinder and obturator. The rubber tipped obturator consists of a slim metal bar about 45 centimetres (eighteen inches) long, one end of which fits tightly into a rubber cork which will just pass comfortably along the rectal cylinder.

The ureter is prepared in the same way and the silk suture is tied to the loop on the end of the punch shaft. A tunnel is formed of requisite length. The rectal cylinder is passed along the bowel, the blunt obturator is withdrawn and the rubber tipped one substituted so that the rubber projects just beyond the end of the rectal cylinder at the level of the distal end of the tunnel. The mucosa punch with obturator in place is passed along the tunnel to the distal fundus, where it is brought hard up against the rubber obturator which the assistant is still firmly holding. The punch obturator is withdrawn. It is now quite easy to punch out the small circular piece of mucous membrane which intervenes between the mucosa punch and the rubber obturator. This is done. The rectal cylinder being held unmoved, the rubber tipped obturator is gradually withdrawn, while the punch, kept closely applied to or even buried in it, follows it down the rectal cylinder until the looped end of the punch shaft has almost reached the tunnel. At this point the rectal cylinder is withdrawn and the end of the mucosa punch is seen protruding from the anus. The rest of the procedure is exactly similar to plan C1.

In this modification, where the mucosa is punched out instead of torn through, it is suggested there is less likelihood of the bowel mucous membrane healing over a deficient ureteral papilla. On the other hand, plan C1 is simpler and quicker.

With the Use of Ureteral Catheters.—Ureteral catheters may be used in executing this technique also. The irreversible tunnel method can be used only for implantation into the large bowel beyond the transverse colon. For implantations higher up the closed tunnel method is suitable.

Cleansing the Bowel.

Bowel cleansing treatment both before and during the operation originally claimed a great deal of our attention and we had elaborated a new and thorough routine, but latterly, in most of the last forty or fifty cases, I have been accustomed to do nothing in the matter. Morphine was given half an hour before operation, and this was usually followed by a bowel evacuation. In many cases, however, even this did not take place and hard fæces had, not infrequently, to be milked from the bowel during the operation itself. I could never see that neglect of bowel cleansing made any difference to the success of the operation. This policy of neglect of bowel treatment is not counselled, but my experience convinces me that overmuch importance has been given by many authorities to this chapter.

In the after treatment I endeavoured as far as possible to restrict the diet of the animals to milk and soft foods for the first few days, but not infrequently the animals eluded vigilance and were discovered eating meat and other hard foods very shortly after the operation without apparent deleterious effects.

DISCUSSION.

Complications.

I shall briefly indicate the complications encountered following the operation of uretero-intestinal transplantation and their causes and, later, the measures that have been adopted to combat them. The complications are as follows:

Peritonitis.

Peritonitis may or may not originate from the site of operation. In experimental work peritonitis not originating from the site of transplantation is usually due to infection from the parietal wound which has been opened by the dog itself.

Peritonitis originating from the site of transplantation may be immediate or secondary.

Immediate.—In the Coffey and other operations the bowel lumen is opened directly, and some degree of fæcal contamination is inevitable. Even in Coffey's plan C the transfixing suture traverses the bowel lumen and contaminates the fingers at least.

Secondary.—In the Coffey and similar operations the first row of sutures coapting the cut muscle edges cannot be regarded as completely efficient in retaining the infection which abuts on the inside through the mucosal opening. The second row of sutures then is to be regarded as the ultimate line of defence and since in this layer unbroken peritoneum

is opposed to unbroken peritoneum and fine catgut is of necessity used, the probability is that this union is by no means permanent. The peristaltic muscular action of the bowel must be continually pulling at the second row of sutures to bring the bowel back to normal shape. Furthermore, we have twice observed tonic contraction of the bowel segment which is incised lasting a quarter of an hour and rendering the intestine as hard as whipcord. If this row of sutures gives way prematurely, peritonitis is likely to ensue.

Hydronephrosis and Hydroureter.

Hydronephrosis in these cases is caused by obstruction to the ureter which may lie: (*a*) at any point along the ureter due to kinking, (*b*) along the intramural portion due to pressure of a stitch (the infolding row aggravates this possibility), (*c*) at the cut end of the ureter. If too much ureter protrudes within the bowel, the useless papilla atrophies and the orifice contracts. Of this we have had many examples (compare Protocol 51). Again, if the ureter does not project adequately or at all within the bowel lumen, there is a danger of the perforation in the bowel mucosa healing (compare Protocol 37). Obstruction may be due to some foreign body (for example, shellac of the ureteral catheter in Protocol 42) or ureteral stone (Protocol 19).

Pyelonephritis.

It is pyelonephritis which led Martin[8] to conclude that ureterointestinal implantation would never be a success. After this operation pyelonephritis is due to an ascending infection. The two factors, virulence of the infecting organism and resistance of the tissue, determine its incidence and extent. The organism in these cases is the *Bacillus coli communis*, to which the cut lower end of the ureter is always subjected. Sweet and Stewart[15] have shown that through the lymphatics from here the kidney is directly infected. Young and Davis[16] state:

> It is obvious that in many cases where bacteria reach it through the blood stream, the kidney either does not become infected, or else that the incipient lesions heal quickly and completely, so that no kidney disease can be detected.

We may take it, therefore, that the kidney is always subjected to contact with bacteria in these cases and yet pyelonephritis does not always follow. Resistance of the tissue is then the variable factor. Leaving out of consideration the possibility of preexisting disease, and discounting the probabilities of trauma and diminished blood supply which must be considered equal in all cases, we are left with one circumstance and one which varies from case to case: obstruction to the ureter which, by causing stasis, lowers the resistance of the ureter. In this series of experiments, in all cases of pyelonephritis there has been demonstrable evidence of obstruction, though the contrary does not hold. It is proposed to expand these observations in a subsequent publication.

Prevention of Complications.

The measures adopted to combat complications are as follows:

Peritonitis.—The aseptic irreversible tunnel technique greatly diminishes both immediate and secondary infection. In no case have we seen evidence that infection has grown back along the tunnel. The closed tunnel plan reduces the risk of secondary infection.

Hydronephrosis.—(*a*) Kinking can be avoided by careful selection of implantation site and general principles. (*b*) The absence of stitches and the infolding layer eliminates these as possible causes of obstruction. (*c*) The ureter should project through the mucosal perforation, but not unduly far. If the ureter is split for six millimetres (one-quarter of an inch) I consider eight millimetres (one-third of an inch) projection is adequate. It will be interesting to hear the late results of Coffey's plan C. Further, if the denuded papilla project too far into the fæcal current, acute inflammation causes swelling and diminution of the calibre. These unforeseen accidents cannot be counteracted. Following all these rules, however, the risk of ureteral obstruction and hydronephrosis of variable degree still exists. The cut end of the ureter heals with fibrosis and contracture as a natural corollary. Cutting the ureter obliquely or splitting it helps matters by increasing the perimeter of the contracting margin. What degree of contracture will cause manifestations in the kidney I do not attempt to state, but it is evident that the contracture of healing would be relatively greater in ureters of smaller calibre.

Pyelonephritis.—Believing as I do that in these cases obstruction to the ureter, whether partial or complete, transitory or permanent, is the great factor in the incidence of this complication, for its avoidance I apply the measures enunciated above with the same final observation.

The Use of Ureteral Catheters.

Though the irreversible tunnel operation is well adapted to the use of ureteral catheters, our results with them in seven experiments were bad. Pyelonephritis occurred in two, hydronephrosis in three, pyonephrosis in two, peritonitis in one: due to pressure necrosis of the ureter after six days.

The following we consider the main reason for the bad results in our experiments: so far from maintaining the patency of the ureters and assuring uninterrupted urinary secretion, they actually were a source of obstruction, for the simple reason that they became so often blocked. The presence of a foreign body in the ureter for the space of some days causes trauma and possibly inflammation of the mucosa. Thus blockage is caused by epithelial or other urinary *débris*. Infection would greatly exaggerate the chance of blockage. Another possible factor is buckling of the mucous membrane mentioned by Kirwin.[5] These causes of blockage take increased importance from the minuteness of the bore of catheters which it is necessary to use in small animals like dogs. With

instruments of an adequately large bore they would cease to exist as causes of blockage. Thus in proportion as larger catheters could be used, so would the objection to their use be removed.

BRIEF DESCRIPTIONS OF PROTOCOLS.

Throughout the course of the experiments frequent estimations were made of the function of the implanted kidneys by: (i) blood urea, indican, cholesterol and carbon dioxide combining power estimations; (ii) measurement of and urea content estimations of the rectal urine; (iii) indigo-carmine and phenolsulphonephthalein tests (an intravenous injection was given and the appearance time and concentration noted in the rectal urine by means of a rubber tube in the rectum); (iv) "Uroselectan" pyelography in some cases. These data cannot be given in a paper of this size.

In the first eleven experiments I employed Coffey's technique, either A or B, in ten animals and thirteen operations; Maydl-Moynihan's method was employed with one ureter; the direct method of implantation was employed with four ureters. One animal, number 4, lived six months and died of kidney deficiency due to hydronephrosis and chronic infective nephritis in both kidneys. One ureter had been implanted into the rectum by the Coffey "A" method, the other into the duodenum by the direct method. The others all died shortly after operation, and none of these protocols is reproduced here.

Protocol 12.—On January 22, 1930, left nephrectomy was performed. On January 29, 1930, the right ureter was implanted into the duodenum by the open tunnel method. On February 3, 1930, the animal died from pneumonia. The blood urea reading was high (414 milligrammes *per centum*), though the kidney and implantation area looked satisfactory.

Protocol 13.—On February 5, 1930, cystectomy was performed with simultaneous implantation of both ureters together with the common piece of bladder wall into the rectum. The open tunnel method was employed. This animal died in six days from double hydronephrosis and pyelonephritis.

Protocol 14.—Left nephrectomy had been performed two weeks previously. On February 20, 1930, the right ureter was implanted in the rectum by the open tunnel method. The animal died in eleven days with pyonephrosis.

Protocol 15.—Left nephrectomy had been performed two weeks previously. On February 19, 1930, the right ureter was implanted into the rectum by the open tunnel method. This animal died in twenty-four hours of peritonitis.

Protocol 16.—On February 10, 1930, cystectomy was performed. The left ureter was implanted with a rosette of bladder wall into the rectum; the right ureter was implanted with a rosette of bladder wall into the small bowel. The open tunnel method was used in both cases. This animal died in two months of renal deficiency. There was slight bilateral hydronephrosis, but no pyelonephritis.

Protocol 17.—On February 17, 1930, cystectomy was performed with simultaneous implantation of both cut ureters; the right into the rectum and the left into the ileum. The wound gave way with evisceration of the intestines on the seventh day. No hydronephrosis or pyelonephritis was found at autopsy.

Protocol 18.—Right nephrectomy had been performed two months earlier. On February 25, 1930, the left ureter was implanted into the recto-sigmoid by the open tunnel method. Death followed in six days from localized peritonitis.

Protocol 19.—On February 27, 1930, the right ureter was implanted into the pelvic colon by the open tunnel method. On March 12, 1930, left nephrectomy was

performed. On June 27, 1930, the animal died. At autopsy no peritonitis or adhesions were found. A stone the size of a small pea was found blocking the ureter five centimetres (two inches) from the kidney pelvis. There was also a fragment of stone in the intramural portion of the ureter. The ureter below the large stone was undilated and patent. No papilla was present. The ureter and kidney pelvis were slightly dilated behind the stone and the urine was turbid. The kidney was otherwise normal and showed no evidence of infection.

This implantation would appear to have been a completely successful one. The possibility of a stone in the single kidney was not entertained before death.

Protocol 20.—On March 1, 1930, right nephrectomy was performed. On March 17, 1930, the left ureter was implanted into the rectum by the open tunnel method. The animal died in seven days from a gangrenous wound. No peritonitis was present.

Protocol 21.—On March 1, 1930, right nephrectomy was performed. On March 21, 1930, the left ureter was implanted into the rectum by the open tunnel method. On May 22, 1930, injected phenolsulphonephthalein appeared in the rectal urine in three and three-quarter minutes. On June 13, 1930, the animal died of distemper, the blood urea reading before death being 140 milligrammes *per centum*. At autopsy the kidney was macroscopically normal; neither hydronephrosis nor hydroureter was present. Microscopically there was no evidence of infection or chronic change. There was some evidence of terminal toxic degeneration in the tubules.

Protocol 22.—On March 1, 1930, right nephrectomy was performed. On March 20, 1930, the left ureter was implanted into the rectum by the open tunnel method. The end of the ureter was anchored flush with the mucosal opening. On March 24, 1930, the animal died. At autopsy no peritonitis was present. The implantation area looked healthy and was healed. The ureter did not come through the mucosal opening, and there was partial obstruction at this point. The kidney showed moderate hydronephrosis, but no infection. Both lungs were moderately congested.

Protocol 23.—On March 3, 1930, the right ureter was implanted into the rectum by the open tunnel method. The animal died in six days from peritonitis spreading from an open abdominal wound.

Protocol 24.—On March 5, 1930, the right ureter was implanted into the sigmoid colon by the open tunnel method. On March 20, 1930, left nephrectomy was performed. On May 13, 1930, the dog died of distemper. At autopsy no peritonitis and very slight adhesions were found. The implantation area was satisfactory, the ureter being undilated. The kidney was hypertrophied, but showed no other abnormality. The bowel was not opened because this specimen showed admirably the "cloaca-formation" of the rectum. This rectal dilatation or ampulla was capable of containing at least 200 cubic centimetres of fluid; the dog was a greyhound (see Figure VI).

Protocol 25.—On March 6, 1930, the right ureter was implanted into the rectum by the open tunnel method. Eleven days later the wound opened and omentum protruded. This was repaired. On April 19, 1930, the animal died of pneumonia. Before death the blood urea reading was 30 milligrammes *per centum*. At autopsy no infection or hydronephrosis of the kidney was evident.

Protocol 26.—On March 8, 1930, the right ureter was implanted into the rectum by the open tunnel method. On March 24, 1930, left nephrectomy was performed. At the present date, July 13, 1931, the dog is still in perfect condition. The blood urea reading is generally between 50 and 60 milligrammes *per centum*. The rectal fluid urea content is 2%. Injected indigo-carmine appears in the rectal fluid in three and a half minutes. "Uroselectan" pyelography on August 22, 1930, showed the kidney to be normal.

Protocol 27.—On March 8, 1930, the right ureter was implanted into the rectum by the open tunnel method. On March 24, 1930, left nephrectomy was performed. On April 8, 1930, after being markedly "asthmatic" for over a week, the animal died.

Post mortem the blood urea reading was 200 milligrammes *per centum*. At autopsy the implantation area was seen to be satisfactory. There was no dilatation of either ureter or kidney, no obstruction to the ureter and no evidence of infection in the kidney. The heart showed concentric hypertrophy, but was not examined microscopically. The lungs were normal. This was an old dog with very worn teeth.

Protocol 28.—On March 26, 1930, the right ureter was implanted into the sigmoid by the open tunnel method. On May 28, 1930, the left ureter was implanted

FIGURE VI. This specimen was obtained from the animal of Protocol 24. This specimen shows very strikingly the "cloacal" distension of the lower bowel from the site of implantation downwards.

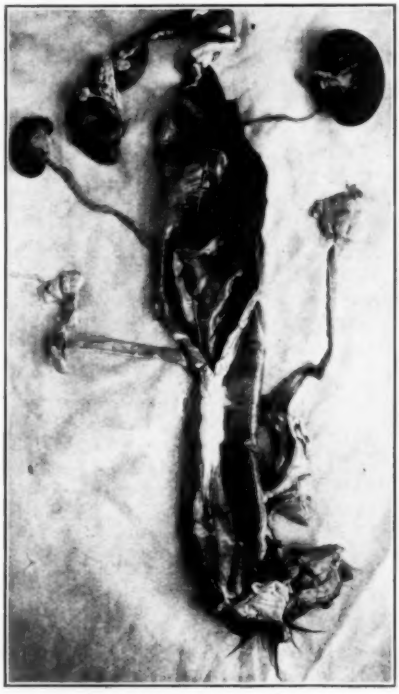

FIGURE VII. Showing the specimen obtained from the animal of Protocol 30. The dilatation or "cloacal" condition of the bowel from the upper implantation site to the anus is notable in this case. The bicornuate uterus and atrophied bladder can be seen in this photograph.

into the rectum by the closed tunnel method. At the present date, July 13, 1931, this animal is still in perfect health. The blood urea readings vary from 40 to 60 milligrammes *per centum*. The rectal fluid urea content is 2%. Injected indigocarmine or phenolsulphonephthalein appears in the rectum in four minutes.

Protocol 29.—On March 28, 1930, the right ureter was implanted into the rectum by the open tunnel method. On April 1, 1930, the animal died following evisceration of the intestines through the wound. No hydronephrosis, hydroureter or pyelonephritis was found at autopsy.

Protocol 30 (see Figure VII).—On March 31, 1930, the right ureter was implanted into the rectum by the open tunnel method. On June 6, 1930, the left ureter was implanted lower in the rectum by the closed tunnel method. Up till March 30, 1931, the animal had always been in excellent condition. Blood urea readings varied from 40 to 90 milligrammes *per centum*. The rectal fluid urea content was 2%. Injected indigo-carmine appeared in the rectum in four and a half minutes. On the date mentioned the dog was killed in the course of another experiment by urea poisoning. At autopsy both implanted ureters were functioning well. Macroscopically the right kidney was normal. Microscopical examination showed some glomeruli adherent in parts to the capsule, otherwise the section was entirely normal. The left kidney showed fibrosis at the upper pole; otherwise it was small but macroscopically normal. Microscopical examination of a section from the lower pole revealed a small area of fibrosis. Some glomeruli were adherent, others, as well as the rest of the section, were normal. A section from the upper pole showed complete fibrosis with shrunken glomeruli. No leucocytes were seen.

Protocol 31.—On April 7, 1930, the right ureter was implanted into the rectum by the open tunnel method. On June 16, 1930, left ureter was implanted into the rectum by the closed tunnel method. On June 21, 1930, the animal died. At autopsy purulent pericarditis and right-sided empyema were discovered, but no peritonitis. The implantation areas were satisfactory.

Protocol 32.—On April 9, 1930, the right ureter was implanted into the sigmoid by the open tunnel method. On April 14, 1930, the animal died from peritonitis spreading direct frfom the open abdominal wound. The implantation area was quite sound and satisfactory.

Protocol 33.—On April 9, 1930, the left ureter was implanted into the sigmoid by the open tunnel method. During the operation the left uterine horn being found in the way, was ligated and divided. On June 1, 1930, the animal died after developing distemper. At autopsy left-sided pyosalpinx was discovered in addition. The implantation area was entirely satisfactory.

Protocol 34.—On April 14, 1930, the left ureter was implanted into the rectum by the open tunnel method. On June 16, 1930, the right ureter was implanted into the rectum by the closed tunnel method. Some fluid bowel contents ran back along the tunnel when the director was inserted. On June 19, 1930, the animal died. Both peritonitis and lobar pneumonia existed. The left implantation area was satisfactory with a normal kidney. The right ureter was strongly imbedded and the implantation was apparently satisfactory.

Protocol 35.—On April 23, 1930, the left ureter was implanted into the rectum by the open tunnel method. On May 28, 1930, no urine having been present in the rectum for two weeks, laparotomy was performed. The animal died under anæsthesia. At autopsy a large left hydronephrosis and hydroureter were found. The ureter was obstructed at the proximal end of the tunnel.

In this instance it seemed that the infolding layer of sutures may have obstructed the ureter by compression.

Protocol 36.—On April 28, 1930, the left ureter was implanted into the rectum by the open tunnel method. On May 16, 1930, injected indigo-carmine appeared in the rectum in seven minutes. On May 26, 1930, the animal died of *cancrum oris*. The implantation area was excellent, showing no obstruction or dilatation of the ureter. No infection or dilatation existed in the kidney.

Protocol 37.—On April 28, 1930, the left ureter was implanted into the sigmoid by the open tunnel method. On June 2, 1930, since no fluid had been present in the rectum for the last two weeks, laparotomy was performed. Advanced hydronephrosis and hydroureter were discovered, with blockage at the tip of the ureter. The ureter was opened and patency reestablished. On July 21, 1930, the right ureter was implanted into the rectum by the closed tunnel method, a ureteral catheter being employed. On September 16, 1930, "Uroselectan" pyelography showed a large right hydronephrosis. The left kidney was not seen. On September 24, 1930, the animal was in a moribund condition. The blood urea reading was 440 milligrammes *per centum*. The rectal fluid urea content was 1·14%.

Laparotomy was performed and a large right hydroureter was repaired. On September 26, 1930, the animal died, with a blood urea reading of 600 milligrammes *per centum*. At autopsy the right kidney and ureter were found to be extremely dilated. The blockage at the end of the ureter had been broken through. There were a large left hydronephrosis and a hydroureter full of clear fluid; the rectal mucosa at the end of this ureter had healed over again. No infection was obvious. No ureteral papillæ were present.

FIGURE VIIIA. This is an enlarged photograph of a section of the descending colon with the ureter running in the wall obtained from the animal of Protocol 39. × 8. In cutting the section the ureter lying outside the bowel has been folded back so as to be included in the same section. The bowel has been opened along the mesenteric border and an artifact produced between the ureter and bowel-muscle.

In this instance the ureters probably did not protrude far enough into the bowel.

Protocol 38.—On April 30, 1930, the left ureter was implanted into the rectum by the open tunnel method. On May 9, 1930, the animal died. The wound and adjacent penis were gangrenous. No peritonitis was present. The kidney and implantation area were satisfactory.

Protocol 39.—On April 30, 1930, the left ureter was implanted into the descending colon by the open tunnel method. On June 23, 1930, the right ureter was transplanted into the ileum by the open tunnel method. On June 25, 1930, the animal died of peritonitis. The kidneys were both normal (see Figures VIIIA and VIIIB).

Protocol 40.—On May 5, 1930, the left ureter was implanted into the rectum by the open tunnel method, with no infolding row of sutures. On May 8, 1930, the animal died of peritonitis. On account of experimental variation in technique this protocol is not tabulated.

Protocol 41.—On May 7, 1930, the left ureter was implanted into the rectum by the closed tunnel method. On May 18, 1930, the animal died of distemper. The kidney was macroscopically normal; on microscopical examination early hydronephrosis was found.

Protocol 42.—On May 14, 1930, the left ureter was implanted into the rectum by the closed tunnel method. On July 14, 1930, the right ureter was implanted into the rectum by the irreversible tunnel method, a ureteral catheter being employed. On August 13, 1930, the animal died. The abdomen was full of blood

coming from a ruptured right pyelo-hydronephrosis. The right ureter was blocked in the intramural portion by pieces of shellac off the catheter. The left kidney and ureter were normal. No papilla was present.

Protocol 43.—On May 14, 1930, the right ureter was implanted into the rectum by the closed tunnel method. On May 18, 1930, the animal died of peritonitis. The implantation area was sound.

Protocol 44.—On May 21, 1930, the left ureter was implanted into the rectum by the closed tunnel method. On June 11, 1930, the right ureter was implanted

FIGURE VIIIB. Showing a part of the same section with higher magnification (× 200). Included in the field are the bowel muscle, bowel mucosa and the whole wall of the ureter. The vessel running along the ureter is well seen. It is clear that there is no infection either round or in the ureter and that the ureter is undilated.

into the rectum by the closed tunnel method. On June 13, 1930, the wound gave way and evisceration and death followed. The implantation areas were satisfactory.

Protocol 45.—On May 21, 1930, the left ureter was implanted into the rectum by the closed tunnel method. On June 2, 1930, laparotomy was performed and patency of a large left hydroureter was reestablished. On June 18, 1930, the right ureter was implanted into the rectum by the closed tunnel method. Very extensive adhesions had to be separated. On June 21, 1930, the animal died of peritonitis.

Protocol 46.—On June 18, 1930, simultaneous transplantation of both ureters was effected into the sigmoid colon through two closed tunnels. On June 27, 1930, the animal died. Double hydronephrosis existed, but no peritonitis.

Protocol 47.—On June 25, 1930, simultaneous transplantation of both ureters was effected into the rectum through a single closed tunnel. On June 27, 1930, the animal died of double pneumonia. No peritonitis was present. Both ureters were kinked and would have become hydronephrotic.

Protocols 46 and 47 are not tabulated.

Protocol 48.—On June 30, 1930, the left ureter was implanted into the rectum by the closed tunnel method with the employment of a ureteral catheter. On August 25, 1930, the right ureter was implanted into the pelvic colon by the irreversible tunnel method without a catheter. On September 26, 1930, the animal died. The left kidney was hydronephrotic; the left ureter was closed (healed over) and dilated. They were both filled with pus. The right kidney was normal with no infection; the undilated ureter was patent throughout.

Protocol 49.—On June 30, 1930, left ureter was implanted into the rectum by the closed tunnel method. On July 10, 1930, the right ureter was implanted into the pelvic colon by the closed tunnel method. During the procedure the terminal anchoring suture unfortunately pulled the ureter right through the bowel wall. On July 12, 1930, the animal died of peritonitis.

Protocol 50.—On July 2, 1930, the left ureter was implanted into the rectum by the closed tunnel method, a ureteral catheter being employed. On July 6, 1930, the catheter was removed. On July 8, 1930, the animal died. Pressure necrosis of the ureter was found 2·5 centimetres (one inch) from the pelvis, and general peritonitis had originated from here.

Protocol 51.—On July 7, 1930, the left ureter was implanted into the rectum by the irreversible tunnel method, employing a ureteral catheter. On July 17, 1930, since no fluid was present in the rectum, laparotomy was performed and the obstructed ureter reopened. On September 1, 1930, the right ureter was implanted into the pelvic colon by the irreversible tunnel method without catheter. On September 10, 1930, the left kidney, which was represented by a hydronephrotic shell, was removed. On January 30, 1931, the animal died. The right kidney was hydronephrotic; there was a pin-point ureteral orifice at the tip of a papilla.

Protocol 52.—On July 7, 1930, the left ureter was implanted into the ileum by the closed tunnel method. On July 31, 1930, the right ureter was implanted into the ileum by the closed tunnel method. On August 4, 1930, urine was discovered in the rectum, having urea content of 1·75%. On August 5, 1930, the animal died of peritonitis from an open abdominal wound. The implantation sites were free from peritonitis. Both kidneys and ureters were normal.

Protocol 53.—On July 10, 1930, the left ureter was implanted into the rectum by the irreversible tunnel method with the employment of the ureteral catheter. On August 14, 1930, no urine was found in the rectum. The animal was killed with ether. Marked hydronephrosis and hydroureter were discovered, with imperforated bowel mucosa.

Protocol 54.—On July 14, 1930, the left ureter was implanted into the pelvic colon by the irreversible tunnel method with the catheter. On August 7, 1930, no urine was found in the rectum. Laparotomy was performed with reopening of the obstructed ureter. Urine appeared in the rectum for a few days. On August 18, 1930, no urine was present and left nephrectomy was performed. The kidney was in the state of pyonephrosis. On September 29, 1930, the right ureter was implanted into the pelvic colon by the irreversible tunnel method without a catheter. On November 17, 1930, the animal died. The kidney was hypertrophied; there was no dilatation of the pelvis or ureter. The kidney was scarred apparently as a result of a post-operative attack of acute pyelonephritis.

Protocol 55.—On July 20, 1930, the left ureter was implanted into the rectum by the irreversible tunnel method with a catheter. On July 30, 1930, the animal died of a gangrenous wound. No peritonitis was present. The left kidney was in a state of acute pyelonephritis.

Protocol 56.—On July 24, 1930, the left ureter was implanted into the rectum by the irreversible tunnel method with a catheter. On July 30, 1930, the animal

died of peritonitis from an open abdominal wound. Intense left-sided pyelonephritis was also present.

Protocol 57.—On July 31, 1930, the left ureter was implanted into the rectum by the irreversible tunnel method with a catheter. On August 18, 1930, no fluid was present in the rectum. The right ureter was implanted into the rectum by the irreversible tunnel method without a catheter. On September 1, 1930, left nephrectomy was performed. This revealed a huge hydronephrotic kidney. On October 8, 1930, the animal died. The right kidney, ureter and implantation area were anatomically perfect. Intense enteritis of the small bowel was present. The blood urea reading did not rise before death.

Protocol 58.—On August 6, 1930, the left ureter was implanted into the pelvic colon by the irreversible tunnel method, a rubber plug in the end of the ureter being used. On September 8, 1930, left nephrectomy was performed. This kidney, in a state of pyonephrosis, was large and friable. On September 24, 1930, the right ureter was implanted into the pelvic colon by the irreversible tunnel method. No catheter was employed. Twelve millimetres (half an inch) of ureter were allowed to project within the bowel lumen. On October 8, 1930, the animal died. Obstruction was discovered at the tip of an elongated papilla. Subacute pyelonephritis and early hydronephrosis were present.

Protocol 59.—On August 11, 1930, the left ureter was implanted into the rectum by the irreversible tunnel method. No catheter was employed. On September 2, 1930, the right ureter was implanted into the pelvic colon by the irreversible tunnel method, again without a catheter. This animal lived in perfect health until May 16, 1931. The blood urea readings varied from 60 to 96 milligrammes *per centum*. The rectal fluid urea content varied from 1·62% to 2·34%. Injected indigo-carmine appeared in the rectum in strong concentration in six minutes. On May 16, 1931, the animal was killed by urea poisoning. At autopsy the left implantation was found to be perfect without any papilla. The ureter was normal and the kidney was normal, save for three superficial small abscesses. The right implantation was anatomically correct. The kidney was represented by a small fibrous bag containing pus. The ureter was somewhat thickened and dilated; the orifice was contracted with no papilla.

Protocol 60.—On August 11, 1930, the left ureter was implanted into the rectum by the irreversible tunnel method without a ureteral catheter. The ureter was allowed to project just through the mucosal opening. On September 3, 1930, the rectal urine being very scanty, the abdomen was opened and the ureteral orifice dilated. On November 29, 1930, the rectal fluid urea content was 0·45%. On February 4, 1931, the right ureter was implanted into the pelvic colon by the irreversible tunnel method without a catheter. On February 9, 1931, 225 cubic centimetres of rectal fluid were present in the rectum; it contained urea 1·62%. On February 22, 1931 the animal died. At autopsy the left kidney was found to be shrunken and nodular. The ureter was patent throughout and reduced in size, being now only slightly dilated. The right ureter was obstructed at the proximal end of the tunnel, probably by the stitch. Slight pyelonephritis existed.

Protocol 61.—On August 14, 1930, the left ureter was implanted into the pelvic colon by the irreversible tunnel method without a ureteral catheter. On September 23, 1930, the rectal fluid urea content was 0·75%. On the same date the right ureter was implanted into the pelvic colon by the same technique. On October 11, 1930, the animal died. At autopsy it was found that the left ureter ended in a pin-point orifice at the side of a papilla. Moderate hydroureter and hydronephrosis containing pus existed. The right side was apparently normal, save for pyelitis. No microscopical examination was made. Other organs were not examined.

Protocol 62.—On August 18, 1930, the left ureter was implanted into the pelvic colon by the irreversible tunnel method without a catheter. At the present date, July 15, 1931, the animal is still in excellent condition. Blood urea readings vary from 35 to 85 milligrammes *per centum*. The rectal fluid urea content varies from 1·35% to 1·95%. Injected indigo-carmine appears in the rectum at six minutes.

Protocol 63.—On August 20, 1930, the left ureter was implanted into the pelvic colon by the irreversible tunnel method. On September 22, 1930, the right ureter was implanted into pelvic colon by the same method. On January 27, 1931, the animal died. The left kidney and ureter were macroscopically normal. The right kidney was pyonephrotic. The ureter was dilated, having a pit-point orifice at the end of a papilla. Death was due to absorption from the pyonephrosis.

Protocol 64.—On August 20, 1930, the left ureter was implanted into the pelvic colon by the irreversible tunnel method without a catheter. On September 24, 1930, the right ureter was implanted into the pelvic colon by the same method.

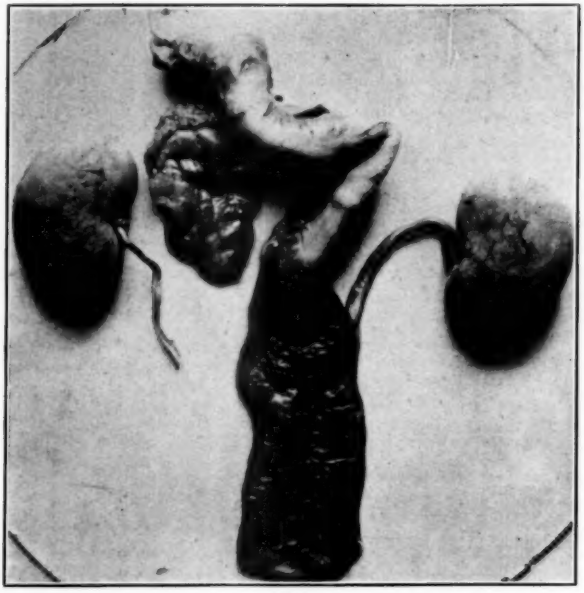

FIGURE IXA. Showing the specimen secured from the animal of Protocol 69. The terminal portion of the pylorus is included in this specimen, the pancreas is also present. The right kidney, whose ureter was implanted into the duodenum for nine and a half months, differs in no way, weight or appearance, from its unimplanted fellow.

On November 10, 1930, the animal died. The left implantation area, kidney and ureter were satisfactory. The right kidney was pyonephrotic; the ureter was dilated with obstruction at the end of an over long papilla.

This protocol should be compared with Protocols 61 and 63; probably septicæmia from the pyonephrotic right kidney caused death.

Protocol 65.—On August 25, 1930, the left ureter was implanted into the pelvic colon by the irreversible tunnel method without a catheter. On September 17, 1930, there being no fluid in the rectum, the abdomen was opened and the patency of the left ureter was reestablished. A large left hydronephrosis was present. On September 22, 1930, the animal was killed by other dogs. A ureteral papilla was present, the orifice of which had been reopened.

Protocol 66.—On August 27, 1930, the left ureter was implanted into the ileum by the closed tunnel method. At the present day (July 15, 1931), the animal is still in excellent health. Blood urea readings vary from 38 to 90 milligrammes *per centum*. There is always urine in the rectum, of which the urea content varies from 0·57% to 2·4%.

Protocol 67.—On August 27, 1930, the left ureter was implanted into the pelvic colon by the irreversible tunnel method without a catheter. On October 2, 1930, the right ureter was implanted into the pelvic colon by the same method. On November 27, 1930, the animal died of pneumonia. The left kidney was hydronephrotic; moderate hydroureter existed with obstruction at its tip. The right implantation area was satisfactory; no infection or obstruction existed.

Protocol 68.—On September 1, 1930, the right ureter was implanted into the duodenum by the closed tunnel method. On September 11, 1930, fluid was found in the rectum, having a urea content of 0·6%. On September 15, 1930, laparotomy was performed. The implantation area looked satisfactory. The ureter was dilated for personal satisfaction. On February 5, 1931, the laparotomy was again performed. The implantation area still looked satisfactory. At the present date (July 15, 1931) the animal is still in perfect health. No fluid has been found in the rectum since September 11, 1930. Blood urea readings vary from 31 to 63 milligrammes *per centum*.

FIGURE IXB. Showing same specimen with kidneys sectioned.

Protocol 69.—On September 3, 1930, the right ureter was implanted into the duodenum by the closed tunnel method. On September 29, 1930, laparotomy was performed. The implantation looked satisfactory, but the ureter was dilated for personal satisfaction. On January 29, 1931, laparotomy was again performed. The implantation area still looked satisfactory. On June 15, 1931, the animal died of severe dermatitis and distemper. Blood urea readings varied from 39 to 103 milligrammes *per centum*. The blood urea *post mortem* was 95 milligrammes *per centum*. No fluid appeared in the rectum at any time. The implantation area was quite satisfactory. The kidneys are identical. The left kidney weighed 28·2 grammes; the right kidney weighed 27·9 grammes (see Figures IXA and IXB).

Protocol 70.—On September 10, 1930, the left ureter was implanted into the pelvic colon by the irreversible tunnel method without a catheter. On September 23, 1930, the animal died of hæmorrhage from a gangrenous wound. No peritonitis existed, also no pyelonephritis or hydronephrosis.

Protocol 71.—On September 15, 1930, the left ureter was implanted into the stomach by the closed tunnel method. On February 5, 1931, the animal was killed with ether. The ureter was found to be sharply kinked 2·5 centimetres (one inch) below the kidney. Hydronephrosis and hydroureter existed above this point. No infection was present. The stomach mucosa had healed over the end of the ureter.

Protocol 72.—On October 8, 1930, right nephrectomy was performed. On January 8, 1931, the left ureter was implanted into the transverse colon by the irreversible tunnel method without a catheter. On January 21, 1931, the animal died. The blood urea reading at death was 1,125 milligrammes *per centum*. The kidney was in a state of hydronephrosis.

This implantation was attempted too high.

Protocol 73.—On October 19, 1930, right nephrectomy was performed. On January 15, 1931, the left ureter was implanted into the pelvic colon as high as possible, adjacent to the transverse colon. The irreversible tunnel method was employed without a catheter. On January 20, 1931, the animal died. The blood urea reading then was 860 milligrammes *per centum*. No peritonitis

FIGURE XA. Showing anterior view of specimen secured from the animal of Protocol 75. The kidney and ureter are macroscopically normal. There is a slight adhesive connection between the ureter and the mesentery just before the tunnel entrance. There are no adhesions or other abnormal appearance in the segment of bowel containing the ureter. The shrunken bladder is seen with the two ureteral vestiges. The bowel from caecum to anus was removed at autopsy.

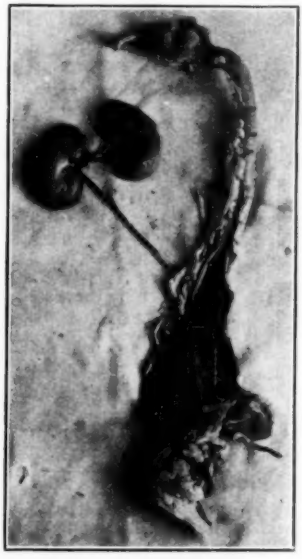

FIGURE XB. Showing posterior view of specimen from animal of Protocol 75. The bowel has been opened along the mesenteric border. The longitudinal ureteral ridge of bowel mucosa is well seen. There is no papilla. An indicator enters the uncontracted ureteral orifice. Flattening of the mucous rugae, giving a cloacal appearance to the rectum, is seen distal to the implantation.

was present. The ureter was sharply kinked 3·75 centimetres (one and a half inches) below the kidney.

This implantation also was attempted too high.

Protocol 74.—On October 16, 1930, right nephrectomy was performed. On January 22, 1931, the left ureter was implanted into the pelvic colon by the irreversible tunnel method without a catheter. On February 11, 1931, injected phenolsulphonephthalein appeared in the rectum in seven minutes. The animal died later in the same day. The cause of death in this case was uncertain.

Protocol 75 (see Figures XA and XB).—On October 8, 1930, right nephrectomy was performed. On January 28, 1931, the left ureter was implanted into the

sigmoid colon by the irreversible tunnel method without a catheter. The end of the ureter was split for 2·5 centimetres (one inch). Part of the split ureter was made to lie within and part beyond the tunnel. On February 9, 1931, the blood urea reading was 141 milligrammes *per centum*. Up till March 24, 1931, the dog had always been in perfect health. Blood urea readings, with one exception,

TABLE I.[1]

Summary of Protocols of Animals Operated on by the Open Tunnel Technique.

Protocols.	Cause of Death.	Lived after Operation.	Protocols.	Cause of Death.	Lived after Operation.
12	Pneumonia	12 days	28	Alive	15½ months
14	Pyonephrosis	11 days	29	Evisceration	4 days
15	Peritonitis	1 day	30	Killed	12 months
18	Peritonitis	6 days	31	Second operation ..	2½ months
19	Ureterolithiasis ..	4 months	32	Peritonitis from wound	5 days
20	Gangrenous wound ..	7 days	33	Distemper and pyosalpinx	2 months
21	Distemper	3 months			
22	Pneumonia	4 days	34	Second operation ..	2 months
23	Peritonitis from wound	6 days	35	Killed	1 month
24	Distemper	2 months	36	*Cancrum oris*	1 month
25	Pneumonia	1½ months	37	Second operation ..	5 months
26	Alive	16 months	38	Gangrenous wound ..	9 days
27	Myocarditis	1 month	39	Second operation ..	1½ months

[1] In this table we have omitted protocols in which simultaneous bilateral transplantations were carried out.

TABLE II.

Summary of Animals Operated on by the Closed Tunnel Technique.

Protocols.	Cause of Death.	Lived after Last Operation.	Protocols.	Cause of Death.	Lived after Last Operation.
28	Alive	15½ months	45 (2)	Peritonitis	3 days
30	Killed	2 months	48 (*c*)	Pyonephrosis	2 months
31	Purulent pericarditis ..	5 days	49 (2)	Peritonitis	3 days
34	Peritonitis and pneumonia	3 days	50 (*c*)	Neurosis of ureter and peritonitis	6 days
37 (*c*)	Hydronephrosis ..	2 months	52 (2)	Peritonitis from wound	5 days
39	Peritonitis	2 days	66	Alive	10½ months
41	Distemper	11 days	68	Alive	10 months
42	Second operation ..	3 months	69	Distemper	9½ months
43	Peritonitis	4 days	71	Killed	5 months
44 (2)	Evisceration	2 days			

(*c*) = Ureteral catheter was employed.
(2) = Two similar transplantation operations were performed.

varied from 28 to 64 milligrammes *per centum*. The rectal fluid urea content varied from 1·11% to 3·06%. Injected phenolsulphonephthalein appeared in the rectum in four minutes. On this date the animal was killed by urea poisoning. The kidney and ureter were perfectly normal. The implantation area was satisfactory. No papilla was present.

Protocol 76.—On October 19, 1930, right nephrectomy was performed. On January 28, 1931, the left ureter was implanted into the sigmoid colon; the same technique was employed as in Protocol 75, with the end of the ureter mechanically dilated. On February 11, 1931, injected phenolsulphonephthalein appeared in the rectum in six minutes. On February 12, 1931, the animal died.

TABLE III.

Summary of Animals Operated on by the Irreversible Tunnel Technique.

Protocols.	Cause of Death.	Lived after Last Operation.	Protocols.	Cause of Death.	Lived after Last Operation.
42 (*c*)	Ruptured pyonephrosis	1 month	61 (2)	Pyonephrosis	2 months
48	Condition of other kidney	1 month	62	Alive	11 months
51 (2) (*c*)	Hydronephrosis ..	5 months	63 (2)	Pyonephrosis	4 months
53 (*c*)	Killed	1 month	64 (2)	Pyonephrosis	2 months
54 (2) (*c*)	Chronic infective nephritis	2 months	65	Killed	5 days
55 (*c*)	Gangrenous wound ..	10 days	67 (2)	Pneumonia	2 months
56 (*c*)	Open wound and peritonitis	6 days	70	Hæmorrhage from wound	13 days
57 (2) (*c*)	Acute enteritis ..	2 months	72	Hydronephrosis ..	13 days
58 (2)	Hydronephrosis and nephritis	14 days	73	Hydronephrosis ..	5 days
59 (2)	Killed	9 months	74	Uncertain	21 days
60 (2)	Hydronephrosis (infected)	18 days	75	Killed	3 months
			76	Pyelonephritis and hydronephrosis	15 days
			77	Pyelonephritis ..	19 days

(*c*) = Ureteral catheter was employed.
(2) = Two similar transplantation operations were perfomed.

TABLE IV.

Showing Results of Operations.[1]

Open Tunnel Method.	*Closed Tunnel Method.*	*Irreversible Tunnel Method.*
25 Operations on 25 Animals.	23 Operations on 19 Animals.	34 Operations on 24 Animals.
8 either are still alive .. 2 were killed .. .. 2 or died following operation on other ureter 4	6 either are still alive .. 3 were killed .. .. 2 or died following operation on other ureter 1	6 either are still alive .. 1 were killed .. .. 4 or died on condition in other kidney .. 1
3 died of pneumonia.	1 died of purulent pericarditis.	1 died of acute enteritis.
1 ,, ureterolithiasis.	2 ,, distemper.	1 ,, pneumonia.
2 ,, distemper.	1 ,, evisceration.	1 ,, hæmorrhage from wound.
1 ,, distemper and pyosalpinx.	1 ,, peritonitis following pressure necrosis of catheter.	1 ,, gangrenous wound.
2 ,, gangrenous wound.	1 ,, peritonitis from open abdominal wound.	1 ,, uncertain cause.
1 ,, evisceration.	5 ,, peritonitis from contamination at operation.	1 ,, peritonitis from open abdominal wound.
1 ,, *cancrum oris*.	1 ,, hydronephrosis.	1 ,, ruptured pyonephrosis.
1 ,, myocarditis.	1 ,, pyonephrosis.	3 ,, chronic hydronephrosis.
2 ,, peritonitis from open abdominal wound.	4 other operations were successful.	2 ,, acute hydronephrosis.
2 ,, peritonitis from contamination at operation.		3 ,, pyonephrosis.
1 ,, pyonephrosis.		2 ,, pyelonephritis with no remarkable hydronephrosis.
		1 ,, chronic infective nephritis.
		10 other operations were successful.

[1] In this table simultaneous bilateral transplantation experiments are omitted.

NOTE.—In the closed tunnel series: The comparatively high incidence of contaminant peritonitis we must assign largely to the practice of leaving the terminal anchoring suture unburied.
In the aseptic irreversible tunnel series: In thirty-four transplantation operations there is not a single instance of peritonitis arising from the implantation site.

TABLE V.

(In this table I have omitted Protocols in which the animals died from the immediate consequence of the operation, that is infected wound and peritonitis, evisceration, hæmorrhage and pneumonia *et cetera*, that is to say animals that died within the first fortnight after operation. Also omitted are Protocols 76 and 77 in which technique was varied. The result on the kidney, that is the success or otherwise of the transplantation operation, alone is considered.)

Method.	Number of Transplantation Operations Concerned.	Apparently Successful.	Hydronephrosis.	Pyonephrosis.	Chronic Infective Nephritis.
Open Tunnel ..	*15*	*13* namely, Protocols 19, 21, 24, 25, 26, 27, 28, 30, 31, 33, 34, 36, 39.	*2* namely, Protocols 35, 37.		
Closed Tunnel ..	*9*	*6* namely, Protocols 28, 30, 42 L, 62, 68, 69.	*2* namely, Protocols 37 (cath.), 71.	*1* namely, Protocol 48 L (cath.).	
Irreversible Tunnel..	*24*	*10* namely, Protocols 48 R, 57 R, 59 R, 61 R, 62, 63 L, 64 L, 67 R, 74, 75.	*7* namely, Protocols 51 L (cath.), 51 R, 53 (cath.), 57 L (cath.), 60 L & R, 67 L.	*6* namely, Protocols 42 R (cath.), 54 L (cath.), 59 R, 61 L, 63 R, 64 R.	*1* namely, Protocol 54 R.
Irreversible Tunnel (omitting Catheter Experiment)	*19*	*10*	*4*	*4*	*1*
Total (omitting Catheter Experiment)	*41*	*29*	*7*	*4*	*1*

NOTE—In seven of the twelve unsuccessful cases the sole fault was blockage of the ureter. In all the others except one obstruction was the main cause of failure. We have pointed out that in larger ureters this factor diminishes.

Moderate pyelonephritis existed. The pelvis was enlarged and contained pus. Probably pyelonephritis was due to excessive trauma and some obstruction to the lower end of the ureter.

Protocol 77.—On February 5, 1931, right nephrectomy was performed. On March 19, 1931, at operation the bladder was removed and the left ureter with a rosette of bladder wall was implanted into the rectum by the irreversible tunnel method without a catheter. On April 2, 1931, the rectal fluid urea content was 1·68%. On April 7, 1931, the animal died. No peritonitis or adhesions existed. Several abscesses were present in the kidney substance, which otherwise had a normal appearance. A well marked ureteral papilla was present with undistensible walls. Very slight, if any, dilatation of the kidney pelvis existed. Acute infective nephritis in the solitary kidney was the cause of death in this and Protocol 76.

SUMMARY.

1. A brief history of the operation is given up to the present date.

2. General surgical opinion judges that the principle of submucous transplantation described by Coffey offers best hope of success.

3. A new technique is described which is truly aseptic. The submucous principle is employed, but the necessity of incision and resuture of the muscle and peritoneum of the bowel wall is obviated.

4. Operations have been carried out on 77 dogs, with the implantation of 106 ureters.

5. Peritonitis as a complication is not serious; none occurred while using the completed technique; that is, 34 operations (one case originating from a gangrenous wound is not considered).

6. Pyelonephritis is not frequent and is always accompanied by some evidence of ureteral obstruction.

7. Hydronephrosis following on contracture of the cut end of the ureter still remains the greatest problem.

8. The use of ureteral catheters, though particularly well suited to the technique, produced bad results in these experiments because they blocked so easily on account of the small bore. In animals in which large catheters could be used, it is considered the objection to their use would be removed.

ACKNOWLEDGEMENTS.

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REFERENCES.

(1) G. Grey Turner: "The Treatment of Congental Defects of the Bladder and Urethra by Transplantation of the Ureters into the Bowel, with a Record of 17 Personal Cases," *The British Journal of Surgery*, Volume XVII, July, 1929, page 114.

(2) C. H. Mayo and W. A. Hendricks: "Exstrophy of Bladder," *Surgery, Gynecology and Obstetrics*, Volume XLIII, 1926, page 129.

(3) R. C. Coffey: "Transplantation of the Ureters into the Large Intestine," *Surgery, Gynecology and Obstetrics*, November, 1928.

(4) H. Wade and D. Band: "Carcinoma of the Cervix Uteri," *The British Medical Journal*, April 11, 1931, page 627.

(5) G. J. Kirwin: "A Study of Ureteral Transplantation," *American Journal of Surgery*, Volume VIII, 1930, page 1.

(6) J. J. Buchanan: "Remote Results of Implantation of the Ureters into the Bowel for Exstrophy: A Consideration of the Extraperitoneal Method of Bergenhem," *Surgery, Gynecology and Obstetrics*, Volume VIII, 1909, page 146.

(7) B. Bergenhem: As quoted by J. J. Buchanan, *loco citato*.

(8) F. H. Martin: "Implantation of the Ureters into the Rectum," *The Journal of the American Medical Association*, Volume XXXII, 1899, page .

(9) R. C. Coffey: "Transplantation of Ureters into the Large Intestine in the Absence of a Functionating Urinary Bladder," *Surgery, Gynecology and Obstetrics*, Volume XXXII, May, 1921.

(10) C. H. Mayo: "Exstrophy of the Bladder," "Osler Memorial Volumes," 1919.

(11) R. C. Coffey: "Completed Aseptic Technique for the Implantation of the Ureter into the Large Bowel," *Surgery Gynecology and Obstetrics*, Volume XLV, December, 1927.

(12) R. C. Coffey: "Production of Aseptic Uretero-Enterostomy by Suture Transfixing Ureteral Wall and Intestinal Mucosa," *The Journal of the American Medical Association*, May, 1930.

(13) R. C. Coffey: "Experimental Transplantation of Ureter in which a Transfixion Suture is Used to Complete the Anastomosis," *North-west Medicine*, Volume XXIX, March, 1930.

(14) I. R. Sisk, J. B. Wear and H. A. O'Brien: "Transplantation of the Ureters to the Sigmoid: An Experimental Study in Dogs," *Surgery, Gynecology and Obstetrics*, Volume LII, February, 1931, page 212.

(15) J. L. Sweet and L. F. Stewart: "The Ascending Infection of the Kidneys," *Surgery, Gynecology and Obstetrics*, 1914, Volume XVIII, page 460.

(16) H. H. Young and D. M. Davis: "Practice of Urology," Volume I, page 123.

THE SPASTIC COLON AS A CAUSE OF CHRONIC ABDOMINAL PAIN.

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ONE of the most difficult problems confronting the physician and surgeon is that of chronic or recurrent abdominal pain with little or no objective signs, when it is often essential to attempt, on symptoms alone, a diagnosis between an organic and a functional cause. When one considers the number of people who have undergone abdominal operation without relief of symptoms, it is clear that anything which may throw light on this field of surgery is to be welcomed. Probably the commonest condition for which patients have suffered unnecessary operation is that known as chronic colospasm or spastic colon.

In this paper I propose to deal fully only with those aspects of the condition which are of special interest to the surgeon, such as diagnosis, and to give merely an outline of treatment, which is more the province of the physician.

At the outset I would point out that the difficulties of diagnosis appear to arise from two facts, namely, that when the condition is purely a manifestation of a neurosis it may closely resemble a surgical condition and, secondly, that colospasm may arise reflexly from an organic surgical condition and resemble that of neurotic origin. While the former has received a fair amount of attention in the last few years, the latter has received almost none.

Hawkins, one of the first to study the subject, wrote in 1906 that colonic spasms "are at the present moment particularly worthy of study, owing to the advance of abdominal surgery, not because they are amenable to surgical treatment, but rather because they need protection." That statement is still true, as will be shown later.

ÆTIOLOGY.

Sex and Age.—Most writers give the sex incidence as two or three females to one male, though in my own series of 45 patients, in which, however, only those showing colospasm under X rays are considered, the proportion was 19 females to 26 males. There is no particular age incidence; my cases range from 20 to 67, the average being 41. This is in agreement with other writers.

Physical and Mental Types.—The asthenic type is commonly supposed to proponderate in this condition, and in the muco-membranous

type this is undoubtedly so. In my series, however, a large proportion are of the stout broad sthenic build. Where the habitus is noted, I find only three of my 15 males described as asthenic and only four of 13 females. The majority of patients, however, are nervous, neurasthenic, worrying, depressed or anxious. Consequently we find the condition more commonly among brain workers than among the public hospital class. Some are introspective, noting the effect of every article of diet on their pains and their stools. Their bowels become their world. Others have phobias of cancer and syphilis. About 20% of my patients had asthma, hay fever or other allergic manifestations, either themselves or in their near relatives, thus tending to confirm the suggestion of some that there is a diathesis or family tendency towards plain muscle spasm, in some cases intestinal, in others bronchial *et cetera*. Nevertheless, few or no allergy signs, for example, eosinophilia *et cetera*, are found by those who have specially investigated this aspect of the problem.

Predisposing Conditions.—As would be expected from the pathology of colospasm, anything that increases the activity of the neuro-muscular apparatus of the colon, or produces irritation of the colonic mucosa, must be regarded as a predisposing cause. Those most commonly found are constipation, faulty diet, the abuse of purgatives, dysentery, lead and tobacco, *tabes dorsalis* and such reflex causes as gall stones, renal calculi and the like.

Past History.—As has already been stated, the frequency of past but unsuccessful abdominal operations, especially appendicectomy and occasionally cholecystectomy, strikes the observer at once. Various writers give figures varying from 23% to 35%. In my series 11 patients (24%) had had abdominal operations without benefit. Of these, only three had pain in the right iliac fossa. Three had pain in the left iliac fossa and five in the epigastrium.

Apart from the above, the past history is usually extraordinarily free from other diseases and infections, except, as stated above, nervous breakdowns, asthma, hay fever, migraine *et cetera*. The patients usually belong to long-lived families.

SYMPTOMS.

The symptoms may be described under several headings.

Pain and Discomfort.—Although not all patients complain of pain or discomfort, this is certainly the commonest symptom, and varies from what is described as "a ball," "a lump" or a "bar of lead" to a dull continuous ache, sometimes very severe, gnawing, but never griping or colicky. I have known the pain so severe as to cause unconsciousness. The severity of the pain may lead to diagnosis of gall stone or ureteric colic, or even *angina pectoris*.

Pain may appear anywhere over the abdomen and round to the flanks and even posteriorly (see below).

Whatever its site, it is wider in area than that of peptic ulcer. Diagnosis is complicated by the fact that reflex gastric manifestations appear in the majority of cases. Excluding these, which will be dealt with later, pain due to colospasm may appear at any time and last any time. Sometimes it occurs soon after meals, but more often during those periods when the colon is normally active, that is, at night between 12 and 3 a.m., in the early morning about 5 or 6 a.m., and before breakfast, thus differing from the pain of peptic ulcer. Pain may last from a few minutes to several days, and on some days and not on others. Among aggravating factors we note anything that may increase bowel muscle activity, such as cold and chilling, jolting, worry, purgatives, fruit and hard articles of food, menstruation and often tobacco. Defæcation sometimes eases, but sometimes increases, the pain, or often one finds that defæcation is unsatisfactory; there is a feeling that the bowel has not been properly emptied, a strong desire for defæcation, but meagre results.

The pain, on the other hand, may be eased, often temporarily, by warmth to the abdomen, by hot baths, rest and a holiday free from worry. It is my impression that the prevailing financial depression has produced an increasing crop of colonic pains.

Some patients are free from pain, complaining of nausea or other symptoms only. These mild cases without colic are often overlooked, and treated for hyperchlorhydria, neurasthenia *et cetera*. Three patients in my series had no pain.

Distribution of Pain.—Although pain may be described as appearing over any part of the abdomen, the most common sites, apart from that due to reflex gastric effects, are the right and especially the left iliac fossæ. Other sites are over or under the right and especially the left lower ribs, the flanks, the sacral area, the kidney areas and the hypogastrium. It is obvious, in fact (and to this point I wish to draw particular attention, because no other writer, except Ryle, has done so), that pain is not segmental in distribution. Herein lies much of the difficulty in diagnosis. I have seen no discussion on the question as to why in some diseases pain should be segmental in distribution and in others non-segmental. In considering this, it has appeared to me that the result depends on whether the afferent impulses travel by the sympathetic path (the more usual) or by the parasympathetic (vago-sacral) paths. That the latter is possible is shown by the fact that bladder pain may be felt in the suprapubic region (segmental: sympathetic) or in the sacral area (parasympathetic). In this connexion a case reported by Kinsella, of Melbourne (*The Lancet*, Volume I, 1929, page 1130), is of great importance. His patient, as the result of a fall two years before, had fractured the spine at the level of the third dorsal vertebra with compression of the spinal cord. He had complete paralysis and anæsthesia below the nipple line. His bowels were opened every third day by enema. The bowel movements were always accompanied by colicky pains referred to the abdomen. These painful

stimuli could have been conveyed only by the vagus. The reference of pain to the abdomen must be a purely psychological effect, the brain projecting the pain to the part which it thinks is affected. That this is not necessarily the area of skin nearest the affected portion of bowel is shown in Table I constructed from my series.

TABLE I.

Showing the lack of relationship between the site of the colospasm and the area of pain, in nine cases in which parts of the colon, other than the descending colon alone, were affected.

Site of Colospasm.	Area of Pain.
Ascending colon only.	Epigastrium, right iliac fossa and left iliac fossa.
Transverse colon only.	Epigastrium and left iliac fossa.
Transverse colon only.	Epigastrium only.
Ascending and descending colon.	Epigastrium only.
Transverse and descending colon.	Epigastrium, interscapular region, right iliac fossa and left iliac fossa.
Transverse and descending colon.	Epigastrium and umbilical region.
Transverse and descending colon.	Epigastrium left scapular region, and left iliac fossa.
Transverse and descending colon.	Epigastrium and interscapular region.
Transverse and descending colon.	Epigastrium only.

In the case of hollow organs with a double nerve supply, it is my impression that pain of localized inflammatory origin is reflected *viâ* the sympathetic fibres to the corresponding skin segment, but that when pain or fullness results from increased tension in the walls of the organ (as from spasm) the afferent impulses are carried by the vagus, and the mind more or less correctly refers the pain to the area covering the organ which it has learned by experience to associate with those processes producing such increased tension or fullness. This explanation, however, would have to be varied in the case of certain of the pelvic organs.

The Stools.—Constipation is present in about half of all cases, in others alternate constipation and diarrhœa; some are normal and a few have diarrhœa.

Stools are often unsatisfactory, infrequent, difficult to evacuate and there is a sense of incomplete evacuation. The stools may be small, flattened or fragmented like sheep-droppings. Mucus may be present, but only in a small number of cases. In my own series only three patients had mucus in the stools, apart from one who later developed colitis. Except for the presence of mucus, there is nothing distinctive between the so-called "mucous colitis" and spastic colon, the former probably being merely a more severe stage of the latter and having the same ætiology. "Mucous colitis" is not, of course, a true colitis, the mucosa being quite healthy in appearance, though this condition may develop as a secondary phenomenon.

Reflex Effects.—Spastic colon may give rise to a number of phenomena which must be regarded as reflex. Some have believed that these phenomena are part of a general hypervagotonicity, for example, pylorospasm, vomiting, occasional bronchospasm, bradycardia, low blood pressure *et cetera*.

The careful work of Bockus, Bank and Wilkinson, however, shows that this is not so, and my own test meal results are in agreement with their results. That distant reflex effects can be produced, however, is readily shown in suitable cases by pressure on or attempted distension by air or enema of a spastic colon. The various reflex effects will be dealt with under their appropriate headings.

Gastric Manifestations.—Gastric manifestations appear in the majority of cases as pain, flatulence, nausea or vomiting, alterations of appetite *et cetera*. Epigastric distress appears either as a fullness or discomfort or as a definite pain one to four hours after meals, burning or gnawing, resembling peptic ulcer, but invariably associated with bowel symptoms. Even late pain after food is not always relieved by alkalis or food, thus differing from duodenal ulcer, but is commonly eased by bowel action. These pains are at their worst when the stools are pencil-like and scybalous. They are probably due to reflex pylorospasm. In my series pylorospasm was observed in 33% of all cases in which the stomach condition was noted, but there was no relation between this and the pain time after food (see Table III). Some had

TABLE II.

Showing the situation of the pains and their time relationships in forty-five cases of spastic colon. In many cases two or more of the pains were combined. (The figures are given as percentages.)

Situation of Pain.	Immediately After Meals.	0–1 Hour After Food.	1–4 Hours After Food.	Variable Pain.	Total.
Epigastrium	20	9	22	16	67
Umbilical region ..	0	2	2	2	6
Right iliac fossa and flank	0	0	0	20	20
Left iliac fossa and flank	4	0	0	36	40
Hypogastrium	2	0	0	16	18
Scapular region	7	0	2	9	18
No pain	6%				

TABLE III.

Showing the situation and time of pain in twelve cases in which pylorospasm was shown by X rays to accompany colospasm.

Epigastric pain—	
Immediately after food	5 cases
0–1 hour after food	1 case
1–4 hours after food	2 cases
Variable pain	2 cases
No pain	1 case
Variable hypogastric pain	1 case

pain in the scapular area. Other common gastric symptoms are flatulence, often with much abdominal distension and loosening of clothes, eructations and belching and nausea, sometimes with vomiting, in about half the cases. The majority have a good appetite, but some are markedly the reverse.

Other Symptoms.—Other symptoms frequently noted include headaches, insomnia, frequency of micturition and asthma (probably mani-

festations of the general neuro-muscular irritability), loss of body weight and cardiac and vasomotor disturbances, the last named probably being connected with the emotional instability, depression *et cetera*.

PHYSICAL EXAMINATION.

Examination often reveals very little. The habitus and type have already been described. In most cases, but by no means all, the affected part of the colon can be felt and rolled under the hand as a small firm cylinder, an unduly hard cord, rod or sausage mass, which may be tender, but not always. The degree of hardness varies from time to time. The descending colon and sometimes the transverse colon are the parts most commonly felt. There often appears to be considerable difficulty among observers in determining as to when a colon is to be regarded as in a spastic condition. In many of my cases the colon was not felt and the diagnosis was made from symptoms and X ray findings alone. It is on account of this difficulty that in my series herein presented only those colons shown to be spastic by X rays have been considered, although it has meant the exclusion of a number of cases, about which there was no doubt, because the patients were not examined by X rays. In some cases, of course, the spasm is low in the pelvis and cannot be felt except by digital examination *per rectum*, when the condition may appear as a hard ring, "like cartilage."

Urinary examination fairly commonly reveals a phosphaturia.

GASTRIC ANALYSIS.

The only authors so far as I know who have published the results of test meal examination are Bockus and his associates. Of 45 cases, hyperchlorhydria was found in 14, normal acidity in 18, hypochlorhydria in 11 and achlorhydria in two. There were no significant abnormalities as regards motility. My small series is in contrast with this. Of 11 patients, six had achlorhydria, one had hypochlorhydria, two were normal, and one had hyperchlorhydria. In all but one, emptying was occurring rapidly between one and one and three-quarter hours.

X RAY APPEARANCES.

The barium meal is a much more satisfactory method of showing a colospasm than is the barium enema, since the slow injection of the latter may overcome an existing spasm.

The barium meal, of course, will not reveal all patients subject to colospasm, but only those in whom spasm happens to coincide with the time of the meal, since spasm is an intermittent phenomenon. The commonest findings by this method are as follows.

Stomach.—The tonus and activity of the stomach are usually good. In 37 of my cases in which the stomach condition was noted, two (5%) were hypertonic, 71% orthotonic and 24% hypotonic. In the two hypertonic, hyperperistaltic cases the pain was epigastric, but most variable

in appearance, and both had tenderness and diarrhœa. Pylorospasm was noted in 33%, no doubt accounting for most of the gastric symptoms, though clinically there was nothing characteristic of the condition. Three patients also showed a marked "hypersecretion" under X rays. All of these had epigastric pain, nausea and flatulence, but a good appetite, while gastric analysis revealed low or absent hydrochloric acid and an emptying time of one and a half hours or less.

Small Intestine.—There is in the majority of cases a condition of marked irritability of the small intestine, with rapid progress of the meal, even along the proximal colon.

Large Intestine.—Colospasm is seen chiefly in the transverse and especially the descending colon. The barium in the affected part appears as a thin thread or a series of small beads. Normal haustrations may be obliterated. The length affected may vary from the whole transverse and descending colon down to one or two inches. As a result of distal colospasm there may be stasis and ballooning of the ascending colon. This occurs in about 30% of cases. Coloptosis is present in about half of all cases.

The Gall Bladder.—The gall bladder was examined in 19 of my cases, of which 11 showed normal function; three gall bladders were under suspicion and those of five patients were regarded as having gross dysfunction. Of the last group, two were subjected to cholecystectomy, but with improvement in neither.

DIAGNOSIS.

Space forbids a full discussion on differential diagnosis, and only a few points will be mentioned. The chief conditions that have caused confusion are:

1. Peptic ulcer. In colospasm pain is associated with bowel function, for example, before breakfast. Relief is often not obtained by alkalis and food, except when eructation of gas occurs.

2. Appendicitis. In colospasm usually no pyrexia, muscle rigidity and cutaneous hyperæsthesia occur, but the history, psychology and stools give a clue.

3. Carcinoma of colon may be accompanied by colonic irritability, spasm and pain following the use of cathartics.

4. Diverticulitis may also accompany spastic colon.

5. *Angina pectoris.*

6. Renal and gall stone colic.

7. Ovarian and tubal disease and dysmenorrhœa.

8. Neurasthenia and hypochondriasis are common diagnoses.

9. True colitis.

This list will indicate the necessity for an X ray examination and a search for blood in the stools.

PATHOLOGY.

The actual nature of tonic hardening of plain muscle is a much discussed and ill-understood problem at the present time. The A. V. Hill school holds that it is merely a tetanus in which very slowly relaxing muscle fibres are involved, thereby accounting for the very low metabolism found in muscle during tonic activity. The colon, therefore, appears as a fixation or postural hardening of the muscle, occurring in any position of extension or contraction. When this fixation produces pressure on the contents of the colon, in opposition to its normal tonus or peristaltic activity, pain is experienced, when the rise in pressure is sufficient. The fact that the pain is of a continuous, aching character, and not short, colicky and intermittent, shows that a steady pressure is being exerted on intestinal contents. Peristaltic activity could not do this.

Sherrington has shown that this muscle hardening is under nervous control and is in fact a reflex nervous phenomenon. It is therefore obvious that the causes of the phenomenon are to be found in those factors which facilitate the reflex. These may be briefly summarized as follows:

1. Increased irritability of the neuro-muscular mechanism—
   - (*a*) Overactivity of the parasympathetic system:
     - (i) Of central origin, following inborn or acquired idiosyncrasies, endocrine unbalance *et cetera*.
     - (ii) Of reflex origin from appendicitis, cholecystitis, renal calculus *et cetera*.
   - (*b*) Underactivity of the sympathetic system, for example, mental or physical exhaustion.
2. Irritation by bowel contents, for example, "roughage" in food (pips, fibre, cellulose *et cetera*), the abuse of purgatives, and possibly the products of carbohydrate fermentation.

As already stated, the neuro-muscular irritability of the colon is, in many instances at any rate, merely part of a similar general condition, as shown by the common association with asthma, urinary bladder frequency and pain, spasmodic dysmenorrhœa, vascular spasm as seen in "dead fingers," and the rapid action of stomach and small intestine. I would suggest that the apparent dysfunction of the gall bladder not uncommonly seen in Graham's test (though no disease is found at operation), is also a manifestation of this irritability. Some authors (Stacey Wilson, Alvarez) hold that the vasomotor changes in the brain are a reflex effect from the bowel, and that the mental symptoms depend on these, disappearing when the bowels are emptied or the colospasm disappears. Others, however, would reverse this relationship.

In contrast with other writers, I have included surgical conditions elsewhere in the abdomen as reflex causes of colospasm, though probably only when the right neurological conditions are also present. My reason is that I have seen cases of colospasm clear up on removal of renal stones, a diseased gall bladder, and on replacement of a retroverted

uterus. I have also produced typical colonic pains by pressure on a large gall bladder full of calculi. This, of course, complicates diagnosis, since (*a*) a case of pure colospasm may be wrongly diagnosed as a surgical condition, and (*b*) a condition correctly diagnosed as colospasm may nevertheless be largely due to a surgical condition.

TREATMENT.

Treatment, which is more in the province of the physician, will be but briefly indicated.

During an attack, probably an injection of atropine is best, combined with an enema at a temperature of 43·3° to 49·9° C. (110° to 120° F.) given slowly, with warmth to the whole abdomen. Between attacks, treatment is directed towards, first, reducing the irritability and, second, reeducating in relaxation: (*a*) the colon and (*b*) the general nervous system.

The most important thing is to attack the general condition of neurosis, anxiety or hypochondriasis. I have known a simple explanation combined with simple remedies to work wonders in an apparently severe and prolonged case. On the other hand, I have experienced utter failure in apparently mild cases. It is unwise to give any prognosis. Diet should be of the "smooth" type, that is, ordinary mixed diet, but with avoidance of pips, cellulose, skins, roughage and a minimum of sugar. Purgatives, irrigations and abdominal massage must be avoided, though general massage may be of value for the neurosis and general subnutrition when present.

Of drugs, belladonna is often described as "the sovereign remedy." My experience with it has been disappointing, but this may be because I have usually reserved it for the refractory cases. In most cases I have found "Luminal" in small doses quite sufficient, when combined with other measures, though, if there is marked mental depression, one should try to avoid depressing drugs. When the stomach empties rapidly, with a lively rapid intestine, whether there is achlorhydria or not, the administration of dilute hydrochloric acid in four mil (one fluid drachm) doses is one of the most satisfactory of all measures. With hyperchlorhydria, bismuth powders are often given, but in my experience with only partial benefit as a rule. I rarely use it in these cases.

AN ANALYSIS OF THE AUTHOR'S SERIES OF FORTY-FIVE CASES.

In this series only those patients who have been found to have colospasm by means of the barium meal and X rays have been included. A number of other patients, obviously suffering from colospasm, but not examined by X rays, were omitted. For this reason my series really represents those cases which were more difficult of diagnosis; just the class, in fact, which gives trouble to the surgeon and physician. For the same reason the figures and percentages shown probably differ widely

in some respects from analyses by others. These figures are as follows (percentages are of all cases):

Pain (see Table II).	
Relief by food	20%
Relief by alkali (11 patients had epigastric pain one to four hours after meals; 9 were relieved by food, but only 4 by alkali)	13%
Tenderness along colon or in epigastrium	53%
Nausea or vomiting	38%
Flatulence	40%
Anorexia	33%
Excellent appetite	49%
Constipation	58%
Diarrhœa	13%
Nervousness, worry, irritability *et cetera*	47%
Loss of weight	40%
Mucus with stools	6%
Asthma, hay fever *et cetera* in personal or family history	18%
Past history of unsuccessful operation	24%
Examination by X rays (gastric condition noted in 37 cases only)—	
Hypertonic stomach	5%
Orthotonic stomach	71%
Hypotonic stomach	24%
Gastric hypersecretion	8%
Pylorospasm	33%
Ascending colon affected	4%
Transverse colon affected	15%
Descending colon affected	93%

LITERATURE.

L. F. Barker: *The American Journal of the Medical Sciences*, 1929, Volume CLXXVIII, page 606.

H. L. Bockus, J. Bank and S. A. Wilkinson: *Ibidem*, 1928, Volume CLXXVI, page 813.

J. D. Dunham: *Annals of Internal Medicine*, 1927, Volume I, page 9.

E. L. Eggleston: *The Journal of the American Medical Association*, 1928, Volume XCI, page 2049.

H. Gauss: *Annals of Internal Medicine*, 1930, Volume III, page 1128.

H. P. Hawkins: *The British Medical Journal*, 1906, Volume I, page 65.

A. F. Hurst: "Essays and Addresses on Digestive and Nervous Diseases," 1926.

J. A. Ryle: *The Lancet*, 1928, Volume II, page 1115.

F. M. Smith, G. H. Miller and W. M. Fowler: *The Journal of the American Medical Association*, 1929, Volume XCIII, page 1932.

S. Wilson: "Tonic Hardening of the Colon," 1927.

CANCER OF THE TONGUE.[1]

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In approaching the subject of treatment of cancer of the tongue, I am reminded of a story I heard at the Westminster Hospital Radium Clinic. Mr. Arthur Evans was showing a case of what had been fairly advanced disease and said: "This man before beginning treatment here returned to his doctor in the country with the statement that the London specialist could cure his cancer of the tongue. The doctor said: 'Oh, is that so? Well, you can tell Mr. Evans with my compliments that he is a liar!'"

Now, so far as the mouth was concerned, the busy practitioner was wrong in his emphatic disbelief. For the whole disease had been made to disappear by radium needle implantation and the tongue was left painless, smooth and mobile. But what of the glands? Was the practitioner nearer being wrong in his sweeping assertion then than at the time, years before, when he learned his surgery under Butlin?

We are here to discuss that problem; to endeavour to lighten the darkness in the fight against metastases—no easy task.

When I meet this problem in a cancer case I always ask myself the question: "What particular procedure will give this particular patient longest useful life with the minimum of suffering, including under the heading of suffering the treatment itself?

Thus very late cases, which, by the way, should never be seen for the first time in these propaganda days, are far better left to euthanasia alone. I am strongly opposed to even local tongue implantation in these because it transgresses the rule already given. The discomfort of implantation far outweighs the benefits and, moreover, discredits the treatment, as response to treatment is poor in feeble subjects. And, speaking of discomfort, may I pause to suggest that many busy practitioners, not familiar with the details of technique, even now seem to consider implantation of needles in the tongue and their retention *in situ* for a week as a very painful business. As a fact, as you all know, it is a perfectly easy matter to implant radium in a tongue under a local anæsthetic without pain, though a local anæsthetic is not the best one to use except in small lesions. The intratracheal method permits of good access and also a relaxation of the tongue muscles so essential for accurate recognition of the extent of the disease. Nor is there any great difficulty in most patients in tolerating the needles for the requisite period. I stress this

[1] Read at the Second Cancer Conference, Canberra, March, 1931.

because several patients have been warned incorrectly about the pain due to the needles.

NEEDLE IMPLANTATION.

It is impossible within the limits of a short address to go fully into the question of implantation technique. I take it that we are all agreed that haste in commencing treatment is necessary; but there should be enough delay to permit of a thorough mouth toilet; a few days will be well spent in attacking septic teeth, clearing up pyorrhœal pockets and so forth, but ruthless extraction of sound teeth, especially in proximity to marginal cancer lesions, seems unwise.

In considering the implantation itself I feel that surgeons who use radium should cultivate an accurate three dimension sense. The extent and form of the lesion should be visualized by careful examination made several times. It is a good plan to make exact measurements and thus to construct a plasticine model of the tongue and of the lesion and at leisure to consider methods of needle arrangement suitable to the case. Hasty and unconsidered implantation is bad practice. It makes an excellent lesson to arrange the needles in space suitably supported and to place in and around the constructed model strips of photographic film (wrapped in light-tight paper). These are exposed for uniform periods and then developed and fixed with careful control of conditions. Finally photometric measurement of the density demonstrates the intensity of field at each part of the model. In fact, in difficult cases various arrangements may be built up and tested in this way with resultant benefit in future problems. It will be found that some extensive lesions will require needles in the lesion itself as well as in the healthy tissue around; others can only be uniformly irradiated by a combination of implantation and surface or rather "near distance" irradiation brought about by the use of dental apparatus.

Thus each worker will build up his own methods of producing uniform fields lethal to the cancer cell. He will have a technique more or less his own, which is a summary of what he has seen done *plus* his own experience.

NEEDLE FIXATION.

So also in the matter of needle fixation. The device of implanting needles in pairs point towards point on opposite sides of the lesion and uniting the eyes by a thread lying taut across the lesion is most reliable. Occasionally two needles on one side may be tied to one on the opposite side. Very short needles tend to extrude and with these the uniting thread must be passed on a round needle down the radium needle track and brought to the surface nearer the lesion and then tied to its companion thread opposite similarly treated. Unruly single needles may be kept in place by passing through the eye a separate fine gut thread, both ends of which are carried down the needle track and then brought to the surface in separate places close together and tied across the isthmus of tissue between. This draws the needle well

into its track, buries the eye and is quite positive in action. In this case the strong security thread, also in the eye, acts as a guide only.

DURATION OF IMPLANTATION.

As to the duration of implantation, one's own needles are of a linear intensity such as to produce a lethal field in one week when placed at or about one centimetre apart. Adult types of tumour may with wisdom be given a longer time, up to ten days, and in large lesions it will be found a good plan to rearrange the position of the needles at half-time, with resulting nearer approach to absolute uniformity of field.

I am a believer in generous screenage, and like 0·6 millimetre of platinum or more. When dental apparatus is used, sheet lead is incorporated, the total screenage being brought up to one millimetre of platinum equivalent, not of course for protection (for that would give none), but to permit of a harder γ ray with a less vicious reaction.

With our present knowledge certainty beforehand as to the radio-sensitivity of any one lesion seems impossible. In a general way, therefore, one must sail near the wind in dosage in aiming at certainty of cure. Occasional over-action up to actual burn will occur even with all care in unduly sensitive subjects, and, moreover, an occasional lesion will not quite resolve after an apparently suitable dosage. One must wait patiently in these, as resolution is sometimes delayed, especially in the largish tumiform types. If in two months induration persists, excision with the diathermy knife seems wise. Biopsy is then possible, and the usual finding is of fibrous tissue. Thus is anxiety allayed. In a general way it serves to remember that tumours of the anterior half of the tongue tend to be adult in type and to be more radio-resistant than those occurring in the base of the tongue, which tend towards the embryonal type and to radio-sensitivity.

SURGERY OF ACCESS.

In gaining access to permit of intelligent implantation into lesions of the base of the tongue, I can recommend pharyngotomy after the manner of Trotter, though not so extensive, as excision is not aimed at. I recommend making the incision down to mucosa as a preliminary a few days before implantation and a tracheotomy is almost a necessity during the retention of the needles. I have once very thoroughly implanted radium needles into the whole base of the tongue from side to side under anæsthesia produced by the tracheal route and mirrored guidance, hooking the needles forwards somewhat and anchoring them by opponents thrust backwards in healthy tongue. The rear needles are extracted by grasping the security thread near the eye and pushing it well down the pharynx until the needle is disengaged. In tumours of the base it is wise to carry out implantation in a far larger area than possibly necessary in order to be sure of doing enough. I realize that some of the foregoing makes the remedy seem worse than the

disease, but one is buoyed up with the thought of the radio-sensitivity of such lesions. No general rule can apply. The patient's own psychology is a definite factor. I cannot forget my experience of a well advanced cancer of the pyriform fossa with glands in the neck as large as hen's eggs on the affected side. The patient was sixty years of age, but determined to have treatment. His crateriform lesion was shown by lateral pharyngotomy performed on the sound side to extend well into the neck tissues and had involved the arytenoid eminence. Needles for the primary growth were implanted through this wound, and the neck glands were simultaneously treated by a combination of implantation and radium pack. He also submitted to tracheotomy and to gastrostomy. His passage was stormy, but he recovered. No sign of the disease can be discovered a year later, and he is a very well and active man again. The key to the success was, of course, the radio-sensitivity, and one wonders whether, with such sensitivity, deep therapy would have accomplished as much with far less toil and suffering.

TREATMENT OF THE GLANDS.

So much then for the tongue itself. Surgery has, in this, definitely given way to radium treatment and, broadly speaking, most patients for whom there is a hope of genuine cure may be relieved of their primary lesions by radium. In considering the treatment of the dreaded lymphatic involvement it might well be asked: "If the tongue itself is so easily managed, why cannot the same treatment be applied to the glands?"

In answering this, one has to remember that the primary lesion and a limited region around it only are turned into a state resembling raw beef, from which the normal tissues of the region emerge cleansed of their scourge. I suppose that an able physicist with unlimited radium at his disposal could devise an apparatus to hold the necessary radium sufficiently far away from the neck and with some hundredweights of lead shielding the rest of the body, to bring about this raw red radium reaction throughout the whole of the suspected area which we should have to postulate as extending from the base of the skull to the clavicle on both sides. But until radium is cheap and plentiful, we must abandon the thought, even if the fear of late radium effects in the shape of necrotic cartilage and so forth did not deter us.

It might be added, however, at this juncture that where behaviour under radium treatment of the primary lesion shows it to be highly sensitive, an opinion confirmed by clinical condition and site, full X ray therapy with the hardest possible rays would appear to be sound treatment. All this is as may be; but what are the underlying principles which should guide our actions in managing the lymphatic area?

I think the first is a classification into those hopefully curable and those hopelessly incurable. Typically the first would be a not large primary lesion with no obvious gland involvement or, say, only one group moderately palpable. Typically the last would be one with the glands enlarged and fixed on one or both sides and the general con-

dition poor. There are, of course, many intermediate cases; in some of these the psychology of the patient or of his friends dominates the situation; and then more or less than the ideal is attempted. In others the general condition is a bar to the theoretically proper treatment, and so on. So that one is really left with the type of case that is hopefully curable.

What to do with the man still full of useful years as a citizen who has had his primary adult type of lesion cured by radium? He knows he has cancer, certainly by inference if not by word of mouth, and is strong enough and courageous enough to do anything asked of him.

I am aware that many surgeons are guided to their next step by the presence or absence of palpable glands. Now if the glands are not enlarged, who can say they are not infected with cancer? I am aware that some few cases prove not to be so. Can anyone pick these cases beforehand? And if he cannot, is he rational in assuming that anyone is free? And, if so, surely the treatment suitable to known infection of glands should apply to all.

Some compromise might be made perhaps in the matter of very early cases in grudgingly admitting that small cancers of the tongue of under two months' duration might be treated expectantly.

Nevertheless, I believe that the next step in the treatment of all hopeful cases should be a Crile type of operation on the affected side. Some surgeons use local anæsthesia; I prefer anæsthesia produced by the intratracheal route, and so indeed would most patients.

Now at biopsy not a few of these primary unsuspected glands prove malignant; others again show minute hæmorrhages that are suspicious, and some again appear free. In these last the French do nothing further, but in the affected cases they have led the world in the use of the radium collar. One's reason for following their lead is that recurrence is so often local in the neck and must be due to scattered cells or to some small infected gland unhappily overlooked. I like to think that scattered cells of cancer or small groups in small glands are more easily affected by radium. I like to imagine them less copiously endowed with their growth-promoting enzyme. And I like to believe that the thinner neck I have created by operation allows of a nearer and better approach of the radium collar to its quest. But whether by operating I have robbed the normal tissues of some of their curative response I do not know. Some day this may be settled and perhaps in a way to decide against operation.

My plan in collar treatment is to wait until the neck has soundly healed and most of the operative œdema has subsided. This is usually about a month. I then make a plaster of Paris mould of the front of the neck, carried well behind the mid-lateral aspects, but open enough at the back to allow of removal. It rests on the clavicular and sternal regions and turns up somewhat over the chin. I then take sheet lead one millimetre thick and hammer it with a plumber's tool so that it lines the

whole cast. The lead is carefully lifted out and covered on both sides with adhesive plaster. On the outer side of the lead mould radium needles are so disposed and stuck in place by more plaster as to create a uniform field within the collar. The number of needles used depends upon the size of the neck, and the amount of radium used has varied between 90 and 150 milligrammes. I regard the disposition of the needles as important, and have at times given over a whole evening for this purpose. Here also the photographic test of field intensity is most valuable, and I have been gratified to find how uniform the field is inside this cylinder of origin. It is not a point of origin, but, roughly speaking, a cylindrical surface, and the plain rule of inverse squares from point origin obviously does not apply. The word cylinder implies that the needles are on both sides of the neck and this point is stressed, for it cannot be assumed that the far side is free of cancer infection. The critic will at once say: "If you hope to cure the far side by irradiation, why do you operate at all?" The answer is that rationally I would operate on both sides and, if the glands were palpable, emphatically I would operate, assuming a hopeful case. But it is a big drain on any but the strongest, and better avoided, especially as infection is, if present, less massive and more amenable to irradiation alone.

It should be realized, however, that the two-sided cylindrical arrangement of needles gives a larger port of skin entry even if one side of the neck only were the objective.

The dosage of collar irradiation runs into ten, fifteen and even twenty thousand milligramme hours, and the lead plaster cast is held away from the skin by sponge rubber 1·5 centimetres thick. It is no light task for the patient and may run into ten to fifteen days, allowing for resting periods, and all patients are glad to have it over. It produces general effects of lassitude, anorexia and even vomiting. There are temporary aphonia and loss of taste. The skin becomes reddened and in parts excoriated and at six weeks and two months the parts shrink, become brownish and pliable.

The late radium effects of the collar over a period of years in many clinics have yet to be recorded. One hopes that there will be very few cases of skin and cartilage necrosis.

As regards preventive treatment in carcinoma of the tongue there is still the cry throughout the land for early recognition and treatment. But whilst human nature remains as it is, there will always be some late cases. But this should not stop us from still calling for early diagnosis, and, moreover, we should insist on the importance of precancerous states and their causes in syphilis, tobacco and improper dentures. What should be done with these leucoplakias, plain or heaped up, or even fissured and ulcerated?

An offending tooth should be extracted, the pipe discontinued. And who fails to give potassium iodide in these?

But I feel that unless any of these suspicious lesions fail to clear up, they should be dealt with by a swift and quite easy excision under a

local anæsthetic. One has seen the leucoplakia surrounding a cancer quite disappear when the cancer is treated and cured by implantation of radium, but one can hardly advise irksome radium treatment for pre-cancerous conditions when excision is so easy and effective.

CONCLUSIONS.

1. The surgery of the primary lesion has given way to radium treatment. In massive cases diathermic reduction of size is followed by radium implantation.

2. The lymphatic area is the real problem; classification into hopeful and hopeless cases is useful: (*a*) Highly sensitive types may be treated by deep therapy or collar alone. (*b*) But all good conditioned subjects of early adult type of lesion should have a Crile operation followed by radium collar. (*c*) Advanced cases may be treated palliatively so long as suffering is not greatly added to.

3. The writer has no five-year series to report, but sees promise of a far better five-year survival rate than that of the pure surgical *régime*.

4. The treatment that offers as part of its course a devastating Crile operation calls for improvement and meanwhile we await a good supply of highly selective monochromatic rays or an antidote to the specific growth-promoting substance.

Case Reports.

CIRSOID ANEURYSM OF THE SCALP: SOME OBSERVATIONS ON THE PATHOLOGY AND A REPORT OF A CASE TREATED BY EXCISION.

By Henry Searby,
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Antyllus[1] in the second century was one of the first to record the difference between the signs of an aneurysm which occurs when there is a local dilatation of an artery and that which results from the rupture of an artery. He left a very clear account of the clinical findings which we now know to be present in arterio-venous aneurysm. This term is defined as "any abnormal communication established between the arterial and the venous channels" (Matas). We are apt to think of arterio-venous aneurysms as occurring only in main vessels such as the carotid or the femoral, and not in small and perhaps unnamed branches, nor between arterioles and venules. The elaborate classification, portrayed diagrammatically in text books, serves no useful purpose from the point of view of treatment, which, to be successful, must abolish the communication between the arteries and the veins.

Cirsoid aneurysm, racemose aneurysm, aneurysm by anastomosis (formerly considered to be an anastomosis between arteries) and pulsating angioma are all very probably ætiologically similar to arterio-venous aneurysm, that is, they all probably arise from an abnormal communication between the arterial and the venous systems. Simple angioma may also come into this category. After Antyllus the surgery of aneurysm made no progress until the time of William Hunter, although Albucasius[1] restated, in the tenth century, the views of Antyllus. Until Harvey's time, of course, no progress could be made with the conception of arterio-venous aneurysm, and Hunter[1] first recognized the cause of this condition. But even at the present time statements are found in text books that cirsoid aneurysm, racemose aneurysm, aneurysm by anastomosis and pulsating angioma are true new growths. Reid[2] quotes references to the literature on this subject wherein Wright, in 1831, proved at autopsy that the main mass of pulsating vessels in a cirsoid aneurysm of the scalp consisted of veins, and Wernher, in 1876, observed that excision of the centre of a cirsoid aneurysm caused cessation of the pulsation of the large vessels forming the aneurysm. In 1906 Berry[3] wrote: "It is well known that cirsoid aneurysm of the scalp commonly develops after injury. It seems reasonable to believe that an injury involving both artery and vein would readily allow arterial blood to be transmitted to the interior of the vein. The increased pressure

Figure I. Showing the extent of the aneurysm.

would naturally cause distension of the thin walled veins and a tumour composed of dilated and tortuous veins would be formed."

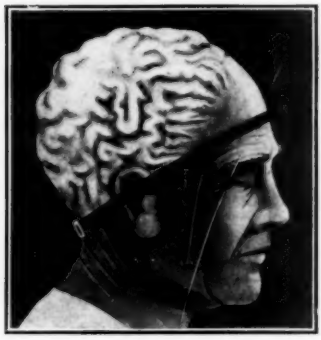

FIGURE II. Showing the position of the tourniquet. (For clarity, in this and the succeeding figures the towels *et cetera* used at the operation have been omitted.)

Reid[2] excised a small but rapidly growing cirsoid aneurysm of the scalp which followed a kick on the head, injected the specimen with a bismuth preparation and then cleared it and demonstrated a very free communication existing between the mass of arteries and veins making up the tumour. He also reports[2] a case of cirsoid aneurysm of the leg in which the anastomosis was partly within the tibia; after amputation the connexion between the arteries and veins could be seen. Some cases of cirsoid aneurysm are undoubtedly congenital and are first seen as nævi, which later increase in size. The development of the capillary bed, an excellent account of which has been given by Florence Sabin,[1] affords countless opportunities for communications between arterioles and venules to persist.

There thus appears to be considerable evidence that cirsoid aneurysm, racemose aneurysm, aneurysm by anastomosis and pulsating angioma should be regarded as due to the same cause as arterio-venous aneurysm; that is, an abnormal communication between the arterial and venous systems and, further, that such abnormal communication may be developmental or traumatic in origin. Because this communication in cirsoid aneurysm is between veins and arteries which, although small, contain blood at a pressure which is high compared with that of the veins, the condition is progressive because the arterial blood in the thin walled veins causes a progressive dilatation. In the case of so-called simple angioma, and of nævi, the difference in pressure between the immediate pre-capillary arterioles and the immediate post-capillary venules is so slight that the vein wall withstands, for the most part, the pressure and a balance is arrived at, so that the condition remains stationary.

FIGURE III. The flap including the galea has been reflected and the communicating vessels tied. The closely packed vessels of the aneurysm are seen cut across. (In this and the following figures the pedicle of the flap has been drawn too narrow.)

Case Report.

Because the journals containing the few references in the last ten years to the treatment of this condition are not available in this country, no attempt is made to discuss the methods adopted by modern surgeons in such cases as that now reported.

An apparently robust labourer, of forty-eight years, was admitted to a medical ward in September, 1928, complaining of increasing dilatation of the scalp vessels for ten years, headaches for two years, gradually becoming worse, and increasing prominence of the eyes. He attributed the condition of his scalp to repeated injuries to his head, as his work for many years had necessitated him lifting heavy cases to and from a lorry. Nothing of importance was elicited in his past history. During the two years prior to admission the headaches had been very severe, lasting for many hours at a time; he had continually felt tired and was sure that his memory was failing.

In bed, except for headache, he felt comfortable, but became cyanosed on the least exertion, although he had no dyspnœa. The whole scalp pulsated with a network of large tortuous vessels forming an elevation of eighteen millimetres (three-quarters of an inch) on the right side of the head and a little less on the left side (see Figure I). To the right of the lambda the dilated vessels formed a mass the size of a large plum and the dilatation extended below the superior nuchal line of the occipital bone into the upper part of the neck. It was impossible to empty the vessels by applying pressure at the various points where normally the main vessels enter the scalp. The temperature was normal, the pulse rate 72 and respiration rate 20. The pupils were equal and reacted briskly to light and accommodation; exophthalmos was present in both eyes, more marked in the left, but the movements of both eyes were normal and painless. The fundi showed early arteriosclerotic changes. The systolic blood pressure was 160 and the diastolic pressure 105 millimetres of mercury. The urine contained no albumin nor sugar, and was clear on microscopical examination. Lumbar puncture revealed clear fluid not under increased pressure, containing a slight excess of globulin, 10 cells per cubic millimetre, 740 milligrammes of chlorides per 100 cubic centimetres, and showing a meningeal curve with colloidal gold. There was no response to the Wassermann test of the blood and of the cerebro-spinal fluid, the blood contained 72 milligrammes of urea per 100 cubic centimetres and the urea concentration of the urine was 3·65%. Skiagrams of the skull revealed no abnormality. Except for an accentuation of the second aortic sound, examination of the heart and lungs revealed no abnormality, as did the examination of the central nervous system.

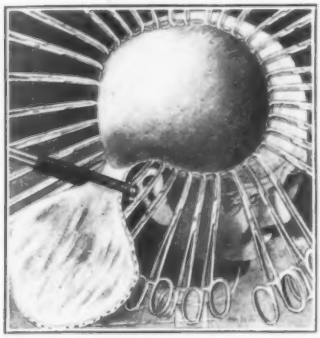

FIGURE IV. The vessels in the flap are controlled by the elastic pressure forceps and those in the scalp by means of closely placed artery forceps grasping the galea. The tourniquet has been removed.

After the patient had rested in bed for a month, the vessels were larger and the headaches controllable only with large doses of morphine. During this time the paranasal sinuses had been investigated as a possible cause of the headaches and the maxillary antra washed out without result. Dr. S. V. Sewell, under whose care the patient was at this time, injected the supraorbital and occipital nerves on the left side with a solution of quinine and urethane, producing analgesia, but no relief of headache. Ten days later the nerves of the right side were injected. The man was then discharged, but had to be readmitted thirteen days later on account of marked increase in symptoms. The headache was even more severe and was now continuous, dyspnœa as well as cyanosis was present on exertion, and the man's mental condition was deteriorating rapidly.

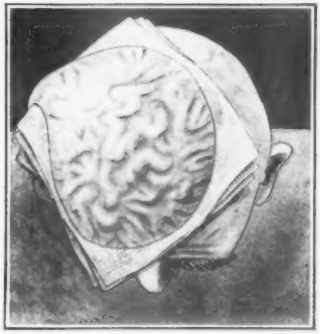

FIGURE V. The vessels have been tied or underrun with silk and the flap replaced with several layers of gauze moistened with normal saline solution between it and the periosteum.

Since a very thorough investigation had thus failed to reveal any condition other than the aneurysm that could account for the symptoms, operation was advised.

First Stage of Operation.

The first stage of the operation was carried out on January 7, 1929. Under ether given by the intratracheal method with the head elevated, a scalp tourniquet was applied tightly across the supraorbital margins in front, over the ears at the side (so as to keep it low enough on the skull) and across the back of the neck at the level of the second cervical vertebra behind (see Figure II). A large flap of scalp, including the galea, was then cut, turned back quickly and an elastic pressure forceps applied across its pedicle. The flap reached to within eighteen millimetres (three-quarters of an inch) of the supraorbital margin in front, almost to the junction of the auricle and the skin of the scalp over the mastoid region on the right side, 3·75 centimetres (one and a half inches) beyond the midline and ended posteriorly in a pedicle 2·5 centimetres (one inch) wide over the lowest part of the occipital bone (see Figure III). The elastic pressure forceps controlled the vessels in the flap satisfactorily and the tourniquet those in the scalp. It could then be seen that the vessels were thin walled, had a diameter about that of an ordinary lead pencil and were packed very closely together. When the flap was turned back some large vessels passing into the skull were divided and tied at once with silk. Curved artery forceps were then placed at intervals of half an inch on the galea and allowed to hang over the cut edge of the scalp tissues (see Figure IV) and the tourniquet removed. One by one the artery forceps were then removed, the individual vessels seized with forceps and tied with silk. Owing to the thinness of the walls of these vessels it was necessary to underrun many of them with silk ligatures on a needle. The individual vessels in the flap were then tied or underrun in a like manner. When hæmostasis was satisfactory, several layers of gauze moistened well with normal saline solution were placed between the flap and the skull (see Figure V), further dressings were placed over the flap, no sutures being used, and the head firmly bandaged. The operation took sixty-five minutes (largely owing to the difficulty in tying the thin walled vessels), and for this reason and because more blood was lost than should have been, the patient was given one pint of citrated blood intravenously. The dressing was not disturbed for seventy-two hours.

FIGURE VI. Appearance of the flap after seventy-two hours. There is some fibrinous deposit on the periosteum; most of the dilated vessels in the flap have thrombosed, but the vessels in the pedicle were patent; the flap is œdematous and the thrombosed vessels and the galea were easily separated from the superficial tissues of the scalp.

Second Stage of Operation.

The second stage of the operation was carried out on January 10, 1929 (seventy-two hours later).

Ether was administered by the open method, and the flap lifted. The tissues of the flap between the skin and the galea were œdematous and about eighteen millimetres (three-quarters of an inch) in thickness. The large vessels in the flap were thrombosed almost to the pedicle (see Figure VI). Owing to the œdema, it was comparatively easy to lift the galea and the thrombosed vessels away from the skin of the scalp in the flap (see Figure VII). At the base of the flap where there was no thrombosis, free hæmorrhage occurred when the œdematous layer of tissue was cut across, but this was easily controlled by underrunning the vessels with silk. The flap was then replaced and anchored at wide intervals with silkworm-gut sutures, two small drains of collapsible rubber tubing being used. This operation occupied twenty minutes.

Progress.

The wound healed by first intention, with the exception of a circular area 1·25 centimetres (half an inch) in diameter, which necrosed at the front end of the flap. This bare area soon healed over. The headaches ceased after the first stage of the operation, although the head was sore. Twenty-four hours after the second stage the patient was free from headache and has remained so since. He returned to his old occupation within a month of his discharge from hospital. The exophthalmos has not quite gone from the left eye yet, but the right eye is normal.

Comment.

It seems certain that the man's symptoms were due to the presence of the aneurysm of the scalp, although many of them are hard to explain. The complete recovery after operation would appear to exclude the possibility that the cirsoid condition was present within the skull. The abnormal vessels forming the aneurysm were between the same layers as are the normal vessels of the scalp. At the second operation most of the tissue containing the normal scalp vessels was excised, but the branches in the outer layers were sufficient to nourish the flap except at the extreme front end. It would have been easier to remove the œdematous tissue if ninety-six hours instead of seventy-two had been allowed to elapse between the two stages; whether this extra time would have rendered the flap more liable to necrose is doubtful, because the thrombosis which occurred was confined largely to the abnormal vessels in the flap and should not theoretically affect the ordinary vessels. It is interesting to note that although only about two-thirds of the aneurysm was excised, the rest disappeared because the abnormal communication between the arterial and venous systems had been abolished.

FIGURE VII. The œdematous mass of tissue, consisting of galea and thrombosed vessels, has been excised and some vessels have been tied near the pedicle. The thin layer of scalp tissue was then replaced and anchored by silk-worm gut sutures.

References.

(1) William Osler: "Remarks on Arterio-venous Aneurysm," *The Lancet*, May 8, 1915, page 949.

(2) M. R. Reid: "Abnormal Arterio-venous Communications, Acquired and Congenital. II," *Archives of Surgery*, May 10, 1925, Volume X, page 998.

(3) W. Berry: "Large Arterio-venous Aneurysm of the Neck Treated by Excision," *The Lancet*, December 22, 1906, Volume II, page 1714.

(4) Florence Sabin: "Reports of Johns Hopkins Hospitals," Monograph, New Series, 1913, Number 5.

ADHERENT PERICARDIUM TREATED BY CARDIOLYSIS.

By EWEN DOWNIE.

[*From the Alfred Hospital, Melbourne.*]

THE following case presents several features of unusual interest. R.O., a male, aged nine years, was admitted to the Austin Hospital, Melbourne, in March, 1930, with myocardial failure of long standing.

The father was confined in a mental home. The mother died in 1922 from burns. One sister, aged twelve years, was a mental defective.

The early history of the boy is unobtainable. For some years he was resident in an orphanage and was admitted to the Children's Hospital, Melbourne, in 1928 with pneumonia. Evidence of old carditis was present at that time. In January, 1929, he was again admitted to the Children's Hospital with myocardial failure which responded to treatment. From July, 1929, until the time of his admission to the Austin Hospital he was confined to bed in the Williamstown Hospital with myocardial failure.

On admission the child was seen to be underdeveloped for his age. The facies was dusky and marked cyanosis of the lips, ears and extremities was apparent.

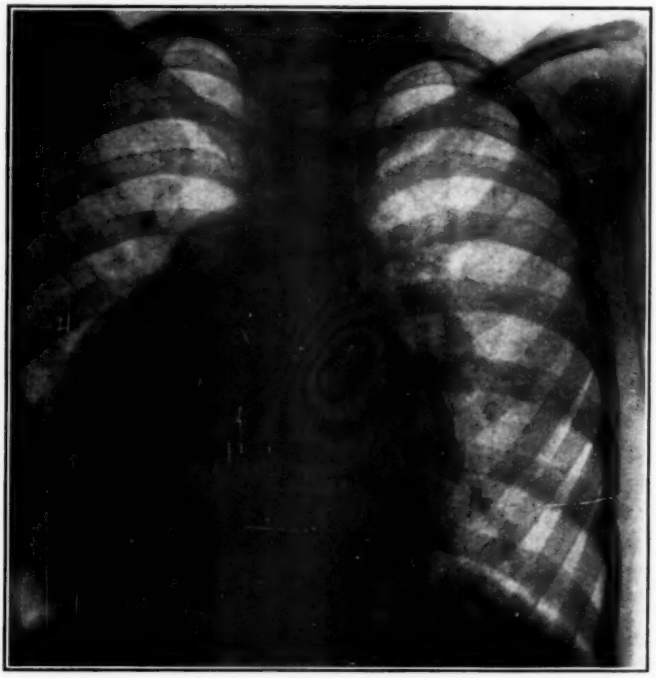

FIGURE I. Skiagram of thorax before operation showing size of the heart seen from behind.

The whole precordial area bulged forwards, and a systolic retraction was visible in the region of the cardiac apex. Diastolic collapse of the veins of the neck was also observed. The heart was greatly enlarged. The apex beat was located in the sixth intercostal space 9·3 centimetres (three and three-quarter inches) from the mid-line. Cardiac dulness was present for 1·8 centimetres (three-quarters of an inch) to the right of the right border of the sternum in the fourth intercostal space. The apex beat was fixed and could not be altered by posture. A loud systolic murmur was present at the apex, replacing the first sound, and was conducted all over the precordium and into the axilla. The second sound at the apex was replaced by a soft diastolic murmur. A soft systolic murmur con-

ducted from the apex was heard at the aortic area; the second aortic sound was clear. The second sound at the pulmonary area was much accentuated. The cardiac rhythm was irregular. The systolic blood pressure was 110 and the diastolic 72 millimetres of mercury. The child did not appear to be distressed whilst lying in bed, and could be exercised without discomfort, but on getting out of bed dyspnœa became apparent with slight exertion.

X ray examination revealed an enormously increased heart shadow (see Figure I) and on screen examination the cardiac excursion was seen to be very limited, the heart scarcely altering in appearance during systole and diastole. An electrocardiogram showed the cardiac irregularity to be due to auricular fibrillation (see Figure II). The Wassermann test yielded no reaction. A diagnosis of adherent pericardium, mitral endocarditis, myocardial failure and auricular fibrillation was made. From the clinical picture it seemed probable that a previous rheumatic infection was the underlying cause.

Under observation it became apparent that the child's condition was largely due to embarrassment of the heart's action by the extensive adherence of the pericardium to the structures of the rigid bony thorax. Accordingly, it was decided to attempt to relieve this by surgical means, and as a preliminary measure to try to lessen the load on the heart by control of the auricular fibrillation.

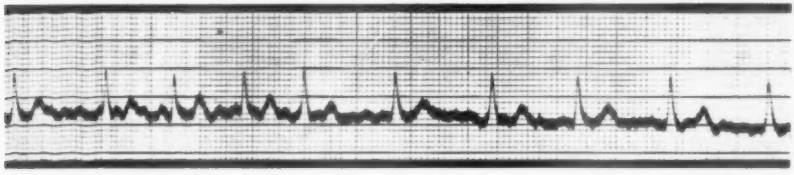

FIGURE II. Electrocardiogram Lead ii showing auricular fibrillation.

On May 18, 1930, the boy was given 0·2 gramme of quinidine sulphate three times per day and this dosage was gradually increased to 0·6 gramme every four hours on May 26. The pulse rate on that day became regular and the quinidine was then discontinued. An electrocardiogram confirmed the fact that the heart had resumed its normal rhythm. No noticeable alteration occurred in the child's condition following the control of the auricular fibrillation.

On June 10 Mr. Hugh Trumble performed the operation of cardiolysis. Briefly, the bony structures of the thoracic wall where the latter was bulged outwards by the enlarged heart were excised. The operation was conducted under local anæsthesia. The intercostal nerves on the left side from the second to the sixth inclusive were blocked with "Novocain" and the skin in the line of the incision was infiltrated. A curved incision was made in the skin over the precordium and portions about 7·5 to 10·0 centimetres (three to four inches) in length of the fourth, fifth and sixth ribs (including the costal cartilages) were excised. The periosteum on the ventral aspect was removed with the ribs, that on the dorsal aspect was not removed. The sternum opposite the ribs excised was freely cut away, together with its periosteum. The retrosternal and retrocostal tissues appeared to be quite elastic and normal in all respects. The area of the thoracic wall thus freed of rigid components was seen to move freely under the influence of the heart beat retracting strongly during systole. The wound in the skin was then closed. Convalescence was uneventful.

The child was kept in bed for ten weeks after the operation. Examination during this time revealed an apparent decrease in the size of the heart, which was later confirmed by X ray examination. The excursion of the heart on screening showed a marked contrast to that seen before operation. In August the child was allowed out of bed and with gradually increasing exercise he was soon able to walk about without distress. By December he was running about, and since that time he has been attending school in the hospital.

There has been no sign of myocardial failure since the operation, and the cyanosis of the lips and the extremities has completely disappeared. The pulse rate varies between 80 and 90 per minute. A slow auricular fibrillation has been present for the past three months.

Discussion.

The surgical relief of pericardio-thoracic adhesions was first suggested by Brauer[1] in 1902. He emphasized the importance of the anchoring of the heart in these cases and suggested that by the removal of appropriate portions of ribs relief of the cardiac embarrassment could be expected. He made no attempt to deal directly with the adhesions. Vaquez[2] subsequently pointed out that, although Brauer's operation was successful in practice, the principles underlying the operation as enunciated by Brauer were incorrect. In Vaquez's opinion the symptoms arise "from the inability of the anterior borders of the lungs to expand during the systolic retraction of the heart, so that the heart, retained by the potential vacuum created around it when it contracts, remains in a state of dilatation. The liberation of the sternocostal structures enables them to be retracted more completely during systole to fill the vacant space, allowing the heart to contract more freely. Thus Brauer's operation, though starting from a mistaken idea, is nevertheless justified by its results." In 1913 Delorme[3] published an account of the operation of pericardiotomy in which he attempted to relieve cardio-pericardiac adhesions by direct division. The operation is not without danger, and is usually followed by the development of fresh adhesions. No such objections exist to Brauer's operation. Bourne,[4] in a review of the literature on this subject, states that the chief indication for cardiolysis is "heart failure of whatever degree accompanied by adherent pericardium."

FIGURE III. Photograph taken after operation showing the alteration in the contour of the thorax.

Such was the indication in the present case. The embarrassment of the heart's action was sufficient to produce considerable deformity of the chest wall during the period of growth. The appearance of the heart on screening showed such a degree of "anchoring" that little change occurred in its size during systole and diastole, with the result that myocardial failure eventually occurred. The reduction which has occurred in the size of the heart since the operation is proof of the obstruction to efficient cardiac action which existed prior to the cardiolysis, and the undoubted benefit which has resulted confirms the indication for surgical intervention.

The occurrence of auricular fibrillation in a child of nine years is worthy of comment, as Cookson[5] has recently drawn attention to the rarity of this condition in the first decade.

Another feature of interest is the tolerance of the child to comparatively large doses of quinidine sulphate.

Summary.

A case of adherent pericardium which was relieved by cardiolysis is described.

The occurrence of auricular fibrillation in a child of nine years and the child's tolerance of large doses of quinidine sulphate are noted.

Acknowledgement.

I am indebted to Mr. Hugh Trumble for his notes of the operation on this case. The accompanying photograph was taken for me by Mr. A. F. Doutch, of the Baker Institute.

References.

(1) L. Brauer: *"Die Kardiolysis und ihre Indicationen," Archiv für Klinische Chirurgie*, 1903-1904, Volume LXXI, page 258.
(2) H. Vaquez: "Diseases of the Heart," First Edition, page 239.
(3) E. Delorme: *"De la légitimité et de l'opportunité de la déstruction des adhérences cardio-péricardiques." Gazette des Hôpitaux de Paris*, 1913, Volume LXXXVI, page 2269.
(4) G. Bourne: "The Operation of Cardiolysis," *Quarterly Journal of Medicine*, 1924, Volume XVII, page 170.
(5) S. Cookson: "Auricular Fibrillation in Children," *The Lancet*, 1929, Volume II, page 1139.

CARCINOMA OF THE JEJUNUM AND ILEUM.

By KEITH ROSS,
Brisbane.

CARCINOMA of the jejunum and ileum is a comparatively rare disease, the clinical course and symptomatology of which are still perhaps sufficiently obscure to render groups of cases worthy of being recorded. This group consists of four cases: one of these occurred in the writer's own hospital practice, two were obtained from the records of the Melbourne Hospital, and one from the private practice of Mr. Victor Hurley, the specimen from this case being in the Melbourne Hospital Museum. In this paper no attempt will be made to deal exhaustively either with the disease or its literature, as excellent papers by Johnson,[1] Rankin and Mayo,[2] and Robb[3] are readily available. Rather will the cases be presented and briefly discussed.

Case Reports.

CASE I.—S.G., a female, aged sixty, was admitted to the Mater Misericordiæ Hospital, Brisbane, on February 16, 1930, suffering from attacks of colicky pain in the abdomen. These attacks had begun four months earlier, and had been preceded for a few weeks by vague abdominal discomfort. From the onset each attack of pain had been severe, situated just below the umbilicus and of several minutes' duration. Attacks had at first occurred at intervals of a few days, but when the patient was admitted to hospital there were several each day. Constipation had become progressively more severe, but there had been no vomiting. A little weight had been lost, but, apart from the distress caused by the pain, the patient had remained in good health.

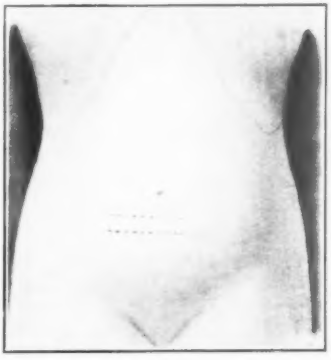

FIGURE I. Showing site of tumour felt in Case I.

When first seen, the patient's abdomen appeared quite normal, but at a subsequent examination during an attack of pain a sausage-shaped tumour, passing transversely across the abdomen below the umbilicus to the right iliac fossa, was felt to come up and alternatively harden and partially relax under the examining hand (see Figure I). The tumour was slightly mobile in a vertical direction, but attempts at movement added to the pain. There was no ladder pattern visible. Radiological examination after a barium meal revealed no abnormality in the gastro-intestinal tract. A diagnosis was made of chronic small bowel obstruction.

Laparotomy revealed a ring carcinoma of the ileum, situated approximately mid-way between the duodenum and caecum. Scattered over the surface of the bowel for 7·5 to 10·0 centimetres (three or four inches) in each direction from the growth were a few small, white, round, flat plaques of secondary growth, and a few more plaques were situated on the surface of the mesentery running to the affected loop of gut and also on adjacent loops of gut (see Figure II). The plaques, numbering in all about twenty, varied in size from 0·2 to 0·5 centimetre in diameter. No enlarged lymph glands could be discovered in the mesentery nor any other secondaries. As it was impossible to excise the growth and its secondaries completely, a lateral anastomosis was performed to short circuit the primary growth. A small plaque was excised for microscopical examination, and revealed an adenocarcinoma extraordinarily faithfully reproducing the glands of the small intestine.

FIGURE II. Showing ring carcinoma and secondaries as seen at operation in Case I.

Immediate convalescence was uneventful, but obstructive symptoms recurred within a few weeks, until finally the patient was readmitted to hospital on June 24, 1930, with complete obstruction. Laparotomy then revealed a gross matting together of numerous loops of small intestine with obstruction at several places, while countless myriads of small miliary secondary growths were thickly scattered over all parts of the parietal and visceral peritoneum. Death occurred two days later. There was no autopsy.

CASE II.—S.W., a female, aged forty-nine, was admitted to the Melbourne Hospital on October 22, 1930, suffering from attacks of colicky pain in the lower abdomen and vomiting. The attacks had begun five months earlier, and, though at first infrequent, had finally become almost daily in occurrence. The appetite was poor, but the bowels were regular. She had lost 22·5 kilograms (fifty pounds) in weight since the onset of the illness.

Examination of the abdomen revealed no abnormality other than slight tenderness in the right hypochondrium. A diagnosis was made of recurring intestinal obstruction.

Laparotomy revealed a mass, apparently carcinomatous, in the ileum, several enlarged lymph glands in the mesentery, but no other secondaries. A lateral anastomosis was performed to exclude the mass. Convalescence was uneventful.

Case III.—C.B., a male, aged seventy, was admitted to the Melbourne Hospital on August 23, 1928, complaining of flatulence and fullness in the upper part of the abdomen. These symptoms had been present for six months, and were relieved by belching and vomiting. The appetite was poor, and the bowels were becoming increasingly constipated. He had had two rigors, the first a month

and the second a week prior to admission. Weight to the extent of 15·75 kilograms (thirty-five pounds) had been lost since the onset of symptoms. He had suffered from gout for many years.

Examination revealed a wasted elderly man with a distended abdomen. The outline of the colon was visible, but abdominal movement was good, and there was no rigidity. Chronic obstruction of the large intestine was diagnosed and an enema produced a good bowel action. Two days later the patient complained of severe pain in the hypogastrium, the abdomen became very distended; coma developed and death ensued on the same day.

An autopsy revealed a constricting carcinoma of the jejunum, 45 centimetres (eighteen inches) from the duodeno-jejunal flexure, with advanced general peri-

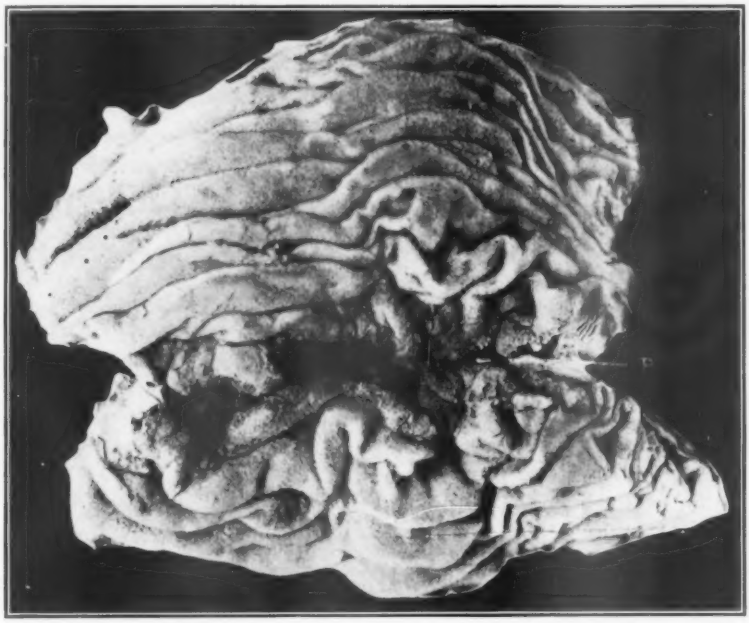

FIGURE III. Showing portion of loop of jejunum excised in Case IV. P marks site of perforation through base of growth.

tonitis due to a perforation through the growth. Numerous peritoneal secondaries were present, situated mostly on the surface of the liver and in the pelvis. Microscopical examination of the growth revealed an adeno-carcinoma.

CASE IV.—C.M., a male, aged fifty-two, who had suffered from a duodenal ulcer for some years, was on April 5, 1930, suddenly seized with acute pain in the upper part of the abdomen and vomiting. On examination he revealed the signs of a general peritonitis, thought to be due to a perforation of his duodenal ulcer.

Immediate operation revealed a perforated carcinoma of the jejunum, 30 centimetres (twelve inches) from the duodeno-jejunal flexure. No secondary growths were apparent. An ulcer scar was present in the first part of the duodenum. The growth was resected and an end-to-end anastomosis of the jejunum was performed. Convalescence was uneventful.

The growth resected is shown in Figure III, and takes the form of a large transverse ulcer completely encircling and constricting the gut. A little fungation is evident in parts. Perforation has occurred through the base of the ulcer. Microscopical examination revealed an adeno-carcinoma.

Comment.

The rarity of the condition is indicated by the fact that the two cases recorded above from the Melbourne Hospital records are the only two that have occurred in that institution between 1922 and 1931, and during that time some 70,000 in-patients have been treated, and autopsies have been held on approximately 75% of the patients who have died in hospital. One case, classified in the hospital records as a carcinoma of the ileum, has been excluded from this series, as there is a little doubt as to the correctness of the diagnosis, while Case II has been included without microscopical verification of the nature of the tumour.

Case I displays most of what may be regarded as the usual symptoms and signs, to wit, flatulent dyspepsia followed by attacks of colic of increasing frequency, vomiting (not present in Case I), progressive constipation, loss of weight, visible or palpable peristalsis and normal findings at a barium meal examination. Case II is similar to Case I.

Cases III and IV are noteworthy in that hospital treatment was not sought until perforation of the growth had occurred, but it is very doubtful if a large series of cases would show such a high incidence of perforation. In Case III suggestive symptoms had been present for some months previously, but in Case IV the patient had either had no preperforation symptoms, or had confused them with those due to his duodenal ulcer.

Two of the growths were situated in the jejunum and two in the ileum, three were of the constricting type and one (unverified) formed a mass in the lumen of the ileum. The two primary tumours and the one secondary submitted to microscopical examination revealed adeno-carcinomatous growth. Cases I and III exhibited gross secondary peritoneal sewing.

Two of the patients were males and two females, and all were in the "cancer period" of life.

Acknowledgements.

I wish to thank Mr. T. E. L. Lambert and Mr. Victor Hurley for their kind permission to publish Cases II and IV respectively, and also Dr. Paul Jones for searching the Melbourne Hospital records for me.

References.

(1) Raymond Johnson: "Carcinoma of the Jejunum and Ileum," *The British Journal of Surgery*, Volume IX, January, 1922, page 422.

(2) F. W. Rankin and Charles Mayo: "Carcinoma of the Small Bowel," *Surgery, Gynecology and Obstetrics*, Volume L, June, 1930, page 939.

(3) J. J. Robb: "Carcinoma of the Ileum," *The British Journal of Surgery*, Volume XIV, January, 1927, page 521.

Reviews.

THE PATHOLOGY OF BONE.

DR. DAVID M. GREIG, the Conservator of the Museum of the Royal College of Surgeons of Edinburgh, has written a book which he calls "Clinical Observations on the Surgical Pathology of Bone."[1] Dr. Greig states that "the surgeon's

[1] "Clinical Observations on the Surgical Pathology of Bone," by D. M. Greig, M.B., C.M., F.R.C.S., F.R.C.S.E.; 1931. Edinburgh: Oliver and Boyd. Demy 4to, pp. 259, with 224 illustrations. Price: 30s. net.

desideratum for his pathology is a working hypothesis which will explain succinctly the processes of disintegration and repair, and if, in his daily work, his observation supports and verifies his hypothesis, so much the better." Dr. Greig is a careful observer and a clear thinker. He endeavours to show that bone is not subject to a special pathology, but conforms to the laws which regulate all tissue indiscriminately. We think he has succeeded.

The author makes no attempt to cover the whole subject of pathology of bone. He starts with a chapter on general considerations and then deals with such conditions as septic osteomyelitis, tuberculosis of bone, the periosteum and syphilis, facial and cranial affections, traumatic osteomata of long bones and joints, carcinoma and finally hyperæmic calcification.

Bone is a living tissue and as such is always changing. It is a connective tissue of similar origin to fibrous tissue and cartilage. Bone cannot be transformed directly into cartilage or cartilage into bone, or either into fibrous tissue or fibrous tissue into either. Osteoblasts are fibroblasts and their only claim to osteoblastic power is that by their death they may and probably do facilitate the transference of calcium from the colloidal condition in which it exists in the blood serum and lymph to the condition in which it is present in bone. Osteoclasts are mere foreign body giant cells helping to absorb dead bone. For the formation of new bone two main factors are necessary, adequate blood supply and adequate supply of calcium. Increase in local blood supply is favoured by the histamine in the cells of all tissues; a frankly pathological reaction is associated with acetylcholine, a normal constituent of the body. The circulation has varying effects upon bone: maintain the circulation within certain limits and bone remains unchanged; produce a definite hyperæmia and bone undergoes rarefaction, decalcification, osteoporosis; restrict the blood supply and bone undergoes consolidation, increased density, osteosclerosis; cut off the blood supply and bone undergoes necrosis. The supply of calcium for new bone formation may be local or general; it is local in most conditions amenable to surgical treatment. It is to the need for local excess of calcium that the surgeon panders when he employs transplantation of bone. The transplant is decalcified and its fibrous elements are reduced to their primordial condition before new bone forms. Heterotopic bone is explained by reduced metabolism in scar tissue, deposition of calcium, liberation of histamine as a result of the irritating action of calcium crystals, increased transudation of serum, a proliferation of fibroblasts, formation of blood vessels; the bone formed is true bone and the scar tissue surrounding it is its periosteum. The periosteum has two functions; it is a limiting membrane and it is a pathway for blood and lymph vessels and nerves. The periosteum is not osteogenic. It is the raising of the periosteum which in many pathological deformations of bone is all important.

Such are the main considerations of the opening chapter. In the rest of the book Dr. Greig applies them to the conditions already mentioned. His contentions are everywhere illustrated by case histories and reproductions of photographs of specimens from the museum of the Royal College of Surgeons of Edinburgh. This book should be studied by all surgeons who deal with bone conditions; they will in turn be interested, instructed and stimulated. In conclusion, a word of praise must be uttered for the excellent way in which both the letterpress and illustrations have been printed.

A TEXT BOOK OF SURGERY.

THE tenth edition of "Modern Surgery," by John Charles Da Costa is a single volume of 1,400 pages[1] The entire work involved in this revision has been undertaken by the author. In his preface to this volume he states that he has purposely

[1] "Modern Surgery, General and Operative," by J. C. Da Costa, M.D., LL.D., F.A.C.S., assisted by B. Lipshutz, M.D., F.A.C.S.; Tenth Edition, 1931. Philadelphia: W. B. Saunders Company. Melbourne: James Little. Royal 8vo., pp. 1404, with 1050 illustrations, some in colours. Price: 60s. net.

omitted any attempt to deal with X ray and radium therapy or the indications for their use, which he feels cannot be satisfactorily dealt with except by the specialist in these forms of therapy. We are sorry to disagree with the author, for he has so satisfactorily dealt with the ever-expanding subject of "modern surgery" that we regret that he has not included a short summary of the present position of radium therapy.

The work involved in the preparation of this new edition and the crowding into one volume of all the existing knowledge is a stupendous task, but the author should feel well satisfied with his achievement. The title "Modern Surgery" is not ill claimed, as all through the book the author quotes references from recent literature. The references are given in the text and not in the form of a bibliography at the end of the book.

Senior medical students should find it a very useful text book and its appeal should not be confined to students; practitioners in general will find it a very useful source of information and help in their everyday practice. This edition, representative of American surgery, together with the recently published thirteenth edition of Rose and Carless, as representative of English surgery, should serve well to show future historians the position of surgery in English speaking countries in 1931.

BRAIN SURGERY.

DR. ERNEST SACHS, of Washington University, has published a most interesting book on the diagnosis and treatment of brain tumours.[1] He has set himself out to show how a brain tumour should be studied rather than to give a complete account of brain tumours. He has succeeded in his endeavour.

The book opens with an account of surgical anatomy and physiology and of methods of examination. In the classification of tumours the author has relied on the work of Penfield and of Cushing and Bailey. The fourth chapter is devoted to general symptoms and signs of increased intracranial pressure, and the fifth to focal signs and symptoms of cerebral tumours. Tumours of the cerebellum, pons and medulla are considered in a separate chapter. Following a chapter on diseases of the pituitary gland is one on the differential diagnosis between brain tumours and other conditions. Operative and post-operative treatment receive ample discussion.

Throughout the book the author illustrates his remarks by case histories; these are well chosen. Unfortunately, like so many other brain surgeons, he gives no general account of his successes and failures—no indication of what may be expected from operation by a skilled surgeon. Is it because the percentage of successes is so small? This may perhaps be regarded as outside the scope of the book. In any case we believe that all who practise brain surgery will study this book; they will find it well worth while. No expense has been spared in the matter of illustrations, which are first class. The bibliography is excellent.

LECTURES IN MEDICAL HISTORY.

"THE History of Medicine," by Dr. Bernard Dawson, is a reprint of a series of seven lectures delivered by the author to the junior students of medicine of the University of Adelaide.[2] In the first lecture he traces the development of

[1] "The Diagnosis and Treatment of Brain Tumors," by E. Sachs, A.B., M.D.; 1931. St. Louis: The C. V. Mosby Company. Crown 4to., pp. 396, with 224 illustrations, including ten in colours. Price: $10.00 net.

[2] "The History of Medicine: A Short Synopsis," by Bernard Dawson, M.D., F.R.C.S.; 1931. London: H. K. Lewis. Sydney: Angus and Robertson. Crown 8vo., pp. 174, with illustrations.

medical and surgical science from its earliest beginnings in the rituals of the priests and the herb knowledge of the witch doctors; and he outlines the medical and surgical attainments of the Egyptians, Babylonians, Assyrians and Jews. These formed the inheritance of the Greeks, whose contribution to medicine from the pre-Hippocratic period to the Greco-Roman period, which ended with the death of Galen, is outlined in the next two lectures. The fourth and fifth lectures deal with the Dark Ages and the Middle Ages, and describe how the Hippocratic tradition and the teaching of Galen were kept alive by little groups of intellectuals in the backwaters of Italy and Spain and in Arabia, until stability returned and universities were established throughout Europe. The Renaissance period is described in the sixth lecture, and brings us to Vesalius, Paré and Harvey. How many of us know that Cæsarean section was performed successfully fifteen times between 1500 and 1549? The last lecture, headed "The Period of Consolidation," sketches briefly some of the many famous workers who lived in the last three hundred years and the discoveries which gave to us the firm foundation of our modern practice.

Medical history has not yet obtained a place in the curriculum of any of our medical schools and the great majority of graduates really know nothing about the subject. The precedent created by Dr. Dawson might be followed wherever medicine is taught. The subject has a practical value; it is absorbingly interesting and, as presented by this book of one hundred and fifty odd pages, need cause no brain fag.

There is a foreword by Professor H. H. Woollard, who is justly proud of having fathered the idea of these lectures. They were entirely voluntary and "all the students attended all the time."

THE CYCLE OF THE HEART.

IN his book, "The Cardiac Cycle," Harrington Sainsbury expresses some unorthodox opinions.[1] He believes that the arterial pulse is due, not to the passage of blood through the vessel, but to a wave resulting from the impulse given to the closed aortic valves by the sudden increase in intraventricular pressure before any blood is forced into the aorta. He declares that the so-called "diastolic" arterial pressure has nothing to do with cardiac diastole.

He discusses the function of the *bulbus aortæ*, and he expounds a theory with regard to the lymphatic circulation.

In some places his argument is lucid, in others it is difficult to follow. His style of writing is unattractive, and his practice of making notes on the text at the foot of the page is at least annoying. Nevertheless, the book is obviously the result of careful thought and observation, and it contains much that should interest both physiologist and clinician. It can be read easily in an hour or so.

ANÆSTHETICS.

A SECOND edition of Charles F. Hadfield's useful little book on anæsthetics is at hand.[2] The various methods of inducing and maintaining general anæsthesia are carefully described. Adequate consideration is given to local, regional and spinal anæsthesia. The work is written for the general practitioner and the student rather than for the specialist in anæsthetics; therefore a description is

[1] "The Cardiac Cycle," by H. Sainsbury, O.B.E., M.D., F.R.C.P.; 1931. Bristol: John Wright and Sons Limited. Demy 8vo., pp. 79, with diagram. Price: 5s. net.

[2] "Practical Anæsthetics for the Student and General Practitioner," by C. F. Hadfield, M.B.E., M.A., M.D.; Second Edition, 1931. London: Baillière, Tindall and Cox. Demy 8vo., pp. 336, with illustrations.

given of only one of the numerous complex machines employed nowadays in the administration of ether. Of "Avertin" the author remarks that it "is a very valuable addition to the anæsthetist's armamentarium, but it will probably only remain so while it is recognised as an adjunct to anæsthesia rather than an anæsthetic in itself." The book can be recommended as providing a sound grounding in the study of anæsthesia and anæsthetics.

ORTHOPÆDICS FOR LAY WORKERS.

"CRIPPLED Children: Their Treatment and Orthopedic Nursing," by Earl D. McBride,[1] is intended to contain information about the subject of cripples, such as would give workers among these patients a good general idea of the deformities and their prevention. For nurses the book does not contain enough detail. One must disagree with some of the statements. For instance, on page 157 the author states: "If one child in the family has the disease [syphilis], it means that all the children and the mother and father have it." Again, on page 224: "The speech [of children with cleft palates] will improve and become nearly normal if the case is successfully operated on within the first year." On page 241 he advises the use of adhesive plaster to control the fragments in supracondylar fracture of the humerus—this might be very dangerous. In Chapter X some very sound advice is given on the subject of splints and braces. On the whole this book can have only a very limited appeal among social workers and nurses.

RADIOLOGY.

AN old lady was once given a dictionary to read in mistake for another book, and when asked whether she liked it, she replied that it was very interesting, but it changed the subject rather frequently. One is left with much the same impression after reading "Recent Advances in Radiology," by Peter Kerley.[2] The title of the book is a misnomer, since the book is more in the nature of a synopsis of radiology. Practically the whole field of X ray diagnosis is covered and a short chapter on therapy is added. The paragraphs are in many cases long and involved and contain a heterogeneous collection of facts. In spite of its shortcomings, however, there is a tremendous amount of information gathered together in this small volume, and while it is not quite elementary enough for the student and too sketchy for the specialist, there are a great number of practitioners interested in radiology who will find it useful.

HISTOLOGY.

DR. E. C. PIETTE has published an interesting text book of histology for medical and dental students.[3] He has given his purely morphological descriptions a physiological background; he rightly states that histology is not purely a morphological science. The plan of the book is conventional, but special chapters on the autonomous nervous system and the reticulo-endothelial system are included. The book might be useful to surgeons for purposes of reference. The illustrations, though mostly borrowed, are well chosen.

[1] "Crippled Children: Their Treatment and Orthopedic Nursing," by E. D. McBride, B.S., M.D., F.A.C.S.; 1931. St. Louis: The C. V. Mosby Company. Royal 8vo., pp. 280, with 159 illustrations. Price: $3.50 net.

[2] "Recent Advances in Radiology," by Peter Kerley, M.B., B.Ch. (N.U.I.), D.M.R.E.; 1931. London: J. and A. Churchill. Sydney: Angus and Robertson. Demy 8vo., pp. 332, with 120 illustrations.

[3] "Textbook of Histology for Medical and Dental Students," by E. C. Piette, M.D.; 1931. Philadelphia: F. A. Davis Company. Royal 8vo., pp. 476, with 277 illustrations, some in colour.

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